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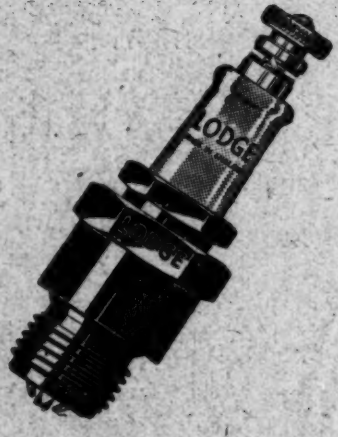

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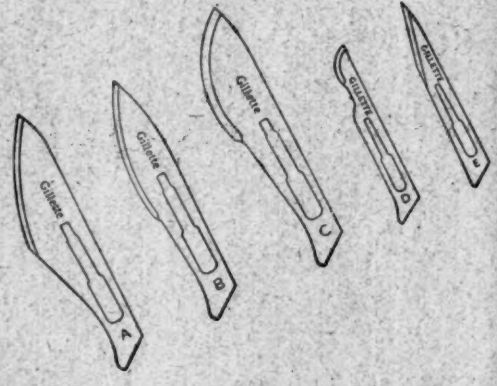

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BRITISH MEDICAL JOURNAL

LONDON SATURDAY DECEMBER 4 1948

"TOWARDS A LOWER PAEDIATRIC MORTALITY"*

BY

WILFRID GAISFORD, M.D., F.R.C.P.

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Although much progress has been made in paediatrics in the past thirty years a great deal yet remains to be done. Repeated reference has been made to the divergence of the curves showing the fall in infant and neonatal mortality rates respectively. There is no doubt about the improvement in the infant mortality rate—a drop from 105 per 1,000 in 1910 to 45.5 per 1,000 in 1944 is certainly significant. Yet for neonates (i.e., in the first month of life) the lag is pronounced—from 38 in 1910 only to 24.5 in 1944. This is partly to be explained by the number of congenital abnormalities which cannot at present be prevented, but also by a big loss of premature infants which could be reduced by stricter attention to antenatal diet and by better immediate post-natal care.

In childhood generally improvement has been steady. Increased interest in paediatrics and especially in prophylactic paediatrics, better nursing care, more health education, smaller families, priorities in food and milk, and the Government issue of free vitamins have all helped. In addition certain specific factors have also helped—for example, the chemotherapeutic drugs and antibiotics, intensive immunization campaigns, and a natural decrease in virulence in certain organisms, notably the scarlet fever streptococcus. Extensions of the immunization programmes may still further improve matters in the future.

Methods of lowering the mortality rate during infancy and childhood may be considered from three aspects—antenatal, neonatal, and post-natal.

Antenatal Paediatrics

The term antenatal paediatrics was introduced by Sir Leonard Parsons in his Blair Bell Lecture to the Royal College of Obstetricians and Gynaecologists in 1945. This lecture provided an immense stimulus to paediatricians throughout the country.

The significance of virus infections in early pregnancy and their relationship to congenital malformations seem now definitely confirmed. The work on the rhesus factor is another example of modern research. From the region supplied by the North-Western Blood Transfusion Service there are approximately 150 Rh-negative pregnant women (with anomalous agglutinins in the serum) every year, and it is hoped to devote a ward specially to the immediate post-natal care of the infants born of these women. The problem may, however, be tackled antenatally—assuming a routine testing to be, as it should, universal. Every female should be tested early in life and careful note taken of those found to be Rh-negative—lest a blood transfusion should ever be necessary and sensitization result from the use of Rh-positive blood, with consequent danger of producing an affected infant from pregnancy in later life. Diamond has shown that such infants are the most severely affected.

Two possible methods of attack have been suggested, both in the experimental stage at present: (1) chemical prevention of the antigen–antibody reaction; and (2) immunological inhibition of antibody formation. T.A.B. injections have been employed for the latter—without success so far; neutralization of maternal antibody has been attempted by Kariher and Miller (1947) using ethylene disulphonate, giving weekly injections during the last half of pregnancy, with suggestively satisfactory results in their first few cases.

There is no doubt that research on these prophylactic lines offers more hope than the present methods of treating the infants after birth, even with replacement transfusions; for nothing can be more disheartening than hours of work spent in transfusing a yellow baby only to find subsequently that a kernicterus already present but unsuspected at birth results ultimately in a mentally defective child. Nevertheless improvement in the prognosis generally has undoubtedly occurred following adequate treatment. The mortality was formerly something over 50%; since the introduction of Rh-negative blood transfusion it has dropped to something like 30%, and with improvements in the technique of replacement transfusions immediately after birth it may be expected to show a further fall.

The Neonatal Period

Prematurity is still one of the major causes of neonatal death, and, although the cause of prematurity is still undiscovered in something like 50% of cases, it is known that *diet* is a matter of outstanding importance in preventing it. Antonov (1947) found that 50% of the births in Leningrad during the siege in 1941 were premature, and that the mothers produced less breast milk and that it lasted for a shorter time. He also noted that mastitis neonatorum—the swelling of the breasts in the newborn that is so common and usually betokens a plentiful milk supply—occurred in only 2% of the infants.

The Australian discovery, reported by Gregg in 1941, of the relationship of German measles in early pregnancy to congenital abnormalities has opened up a large field of inquiry into a hitherto completely obscure subject. The possibility that other infections than rubella—e.g., mumps, influenza, and herpes—may be aetiological is probable, and other deformities than malformation of the heart and eyes may subsequently prove to be related to such infections. If

*Abridged from a lecture given to the Halifax Division of the British Medical Association on March 24, 1948.

allowance be made for the incubation period and for the time taken by the virus to penetrate the placental barrier the actual maternal infection may be placed earlier than the fifth week.

The logical sequel to this knowledge is, of course, that every precaution should be taken to prevent such infections in pregnancy, either by ensuring that they have already taken place earlier in life—e.g., by deliberate exposure in childhood, due care being taken that the child's general condition is satisfactory—or by the rigorous avoidance of exposure during the first three months of pregnancy, or by the administration of gamma globulin if exposure has occurred at this time. Unfortunately the efficacy of gamma globulin in preventing rubella is by no means certain.

The Post-natal Period

It has been generally accepted that newborn infants and infants under three months of age are poor antibody producers and that there is no great point, therefore, in attempts at early active immunization. Recent work by Sako *et al.* (1945) has cast doubts on the truth of the accepted findings, and early immunization may prove to be effective to some extent at any rate. For example, Waddell and L'Engle (1946) immunized 129 infants with pertussis vaccine at one week, one month, and two months after birth, and 70% showed an adequate response. Controls showed no carry-over from the mother.

The site of antibody formation is still a matter of dispute, but if, as Harris and his co-workers (1945) hold, one of the chief sites is the lymphocyte then there is no reason why newborn infants should not produce antibodies, as they normally have 66% of their total white cells in the form of lymphocytes after the first week of life, and gamma globulin is one of the constituents of the lymphocyte. Nevertheless, such immunity takes time to develop. For example, whooping-cough immunity certainly requires two months. Whooping-cough is not common in the first few months of life, yet when it does occur it is fatal in a large percentage of cases (686 infants under a year old died from whooping-cough in 1944 in England and Wales). Diphtheria is also a fatal illness in infants, but it is much rarer.

The idea of immunizing the mother during the later months of pregnancy so that the immunity may be carried over to the infant to last him until such time as active immunization may be capable of carrying on the immunity is well worth considering. It has already been done on a small scale in America by Cohen and Scadron (1943), and was found to be harmless to the mother and child and to result in a reasonable titre in the infant's blood in the first months of life. Similarly, immunization of Schick-positive pregnant women might be a worth-while procedure. Schick-negative women produce Schick-negative infants, who would not need immunizing immediately.

Cohen (1947) has drawn attention to the altered immunity in adult populations as a result of mass inoculations: 85% of adults were immune to diphtheria in 1920—only 50% are now, owing to the loss of artificially acquired immunity from the lessened chances of exposure. This affects the newborn also, who in 50% of cases may thus get no carry-over from the mother. Similarly, a high proportion of adults are not immune to whooping-cough and therefore their babies are susceptible.

Close collaboration between obstetricians and paediatricians is the first requisite in reducing neonatal mortality. We all realize that not every infant can be born at full term, and one of the most important problems is how to deal with premature infants.

Nursing

The most important factor in saving the lives of these infants is nursing. A good gamp in a humble cottage is better than a poor nurse in the most up-to-date "prematorium." As is well known, most premature infants who die do so in the first 48 hours, and very often death occurs shortly after a feed. There is a tendency to atelectasis, the swallowing reflex is poorly developed, and feeds are easily regurgitated and inhaled. Clement Smith, of Boston, gives no fluids at all to premature infants for two to four days after birth. By that time they have got over the shock of birth, their lungs are expanding, and their swallowing reflex is less immature. I have tried this technique for the past six months and the results have been most satisfactory. It is little short of amazing to find that a 2½-lb. (1.13-kg.) baby will go for between 70 and 80 hours without fluids of any sort and yet do very well. Weight loss is not excessive; feeding, when it is started, is a less precarious procedure, and subsequent progress is most satisfactory. Oedema is lost more quickly. The infants are kept warm, given oxygen, and left completely alone except for changing until they indicate by their cry that they are hungry; this crying also expands their lungs. Jaffe (1948) has reported on the progress of infants so treated in the premature station at St. Mary's Hospital.

The question of the advisability of giving vitamin K in all cases, either to the mothers just before labour commences or to the infants at birth, is still debated. Some give it as a routine; others only in cases of difficult delivery where cerebral haemorrhage may be expected; while others reserve it solely for infants who have already bled (i.e., cases of melaena neonatorum). As it is perfectly harmless and it is difficult to judge in any given case whether there will be a hypoprothrombinaemia of pathological intensity or not, it should always be given. Another reason is the possibility of preventing a subdural haematoma from slowly accumulating—giving rise to convulsions later and involving, if diagnosed, surgical evacuation of the clot. Such haematomata are often found at necropsy, and have undoubtedly been the cause of death.

Kerpel-Fronius (1948) has suggested that vitamin P should be given as well; that there are two factors concerned with the diathesis—one the hypoprothrombinaemia and the other an increased capillary fragility—both resulting from vitamin deficiency in the mother's diet. The seasonal incidence has of course been known for a long time, which adds some weight to this theory.

Another frequent cause of death is *infection*. This too is often unrecognized, being rapid both in onset and in spread, and terminating fatally perhaps within 24 hours. Because of the frequency of *Bact. coli* and other Gram-negative organisms—some not generally pathogenic in later life—streptomycin may well come to be the drug of choice in treating such cases. Short intensive treatment is all that is needed, so there should be no question of drug-fastness as in the treatment of tuberculosis.

As the tendency is for more and more women to go into hospital to have their babies the question of nurseries in maternity wards is an interesting and still undecided problem. The advantages of having mother and infant together are obvious, yet comparatively few hospitals make such arrangements. I have made a point of asking mothers in the maternity wards I have visited whether they would like their babies in a cot within reachable distance all the time or whether they would prefer them in a nursery and just brought in for feeds. Their replies have been fairly consistent: the primiparae would like their infants with them; the multiparae prefer a nursery. This is just what is wanted really—the primipara to be taught how to feed and change

and wash her infant, and instructed in the various difficulties to be overcome in breast-feeding, and the multipara to have a well-earned rest. In other words, our new maternity hospitals might well provide special wards for primiparae, each bed having a cot attached, with only a small nursery for the occasional use of a fractious infant who would need to be put out for a night or two; and wards for multiparae as at present; associated with a regular nursery for all their infants.

There is a great need for accommodation for infants born of tuberculous mothers so that they may be removed from "open" contact. The provision of tuberculosis-free foster-homes is the ideal solution: there is less risk of cross-infection than in an institution for babies. Even when B.C.G. vaccination is undertaken on a large scale such accommodation will still be necessary, though for a shorter period of time.

The infant of a diabetic mother may have an urgent need of glucose immediately after birth, and preparations should be on hand to meet this need. Although newborn infants are comparatively insusceptible to hypoglycaemia (blood-sugar levels of 50 mg. per 100 ml. are common in normal infants) the hypertrophy of the islets brought about by the foetal hyperglycaemia may occasionally cause severe symptoms. The very sudden drop in blood sugar which these infants may undergo may easily result in death from hypoglycaemia, yet they may all be saved by prompt treatment. As the infants who manifest these changes are usually large—indeed, over large—the glucose can be given quite adequately by mouth, but if necessary gavage should be used, either intermittently or by continuous drip through a catheter passed into and left in the stomach; or in extreme urgency intravenous injection may be necessary. An essential prophylactic measure is the careful control of the mother's blood sugar during pregnancy.

After the neonatal period the reduction in mortality becomes more and more a preventive programme, the stress being on child health rather than on curative paediatrics; for, though research into curative methods—especially new chemotherapeutic agents and antibiotics—goes on, a distressingly large number of infant and child deaths are due to preventable conditions.

Preventable Diseases

As Davison (1934) has pointed out, of the 100 important diseases which may attack a child 37 are preventable, and these 37 cause more than half the deaths in childhood.

Active immunization has been responsible for a considerable reduction in the death rate in childhood, especially from diphtheria. Every child should have adequate immunization against diphtheria, smallpox, and whooping-cough, and, if he lives in the country, typhoid and tetanus as well. Furthermore, boosting doses should be given at intervals: the rise in antibody titre is both prompt and high after even minute doses—e.g., 0.1 ml. given intradermally. The fact that two or three antigens may be combined with advantage is helpful and reduces the total number of injections necessary: diphtheria and pertussis go well together; so do typhoid and tetanus. When gamma globulin becomes generally available we may have another potent agent against infection.

Deaths from tuberculosis in infancy are still far too numerous. They may be prevented by (*a*) prevention of exposure, as, for example, by the use of foster-homes for infants born into homes where there is "open" tuberculosis; (*b*) by setting up "preventoria" where children suffering from primary infection may be watched till the first three months after their primary infection, which are so vital prognostically, have been successfully passed; (*c*) by B.C.G. or Wells's vole-bacillus vaccination; or (*d*) by not allowing milk that can possibly contain tubercle bacilli to be given to any infant or young child—a point of practical importance which could and should be tackled immediately.

The percentage of cases of tuberculous meningitis which is bovine in origin doubtless varies geographically, but it is by no means negligible—and all could and should be prevented. The Animals' Bill is a big step forward. The Minister of Agriculture reported (*B.M.J.*, 1948, **1**, 371) that even to-day nearly seven-eighths of the cattle population in Great Britain were not attested and that it would take from ten to fifteen years to clear the country of bovine tuberculosis. Till then compulsory adequate pasteurization would help enormously with our problem.

Rickets was a disease which indirectly caused many deaths every year. The fact that a case of florid rickets is now hard to find is a triumph of preventive paediatrics largely due to public-health work and education. The full value of this improvement will not be appreciated till the next generation of infants is born—of women without rachitic pelves.

Advances in Surgery

The amazing advances in cardiac and thoracic surgery provide a contrast: here curative treatment has indeed advanced by leaps and bounds. The names of Blalock and Ladd and Gross have added lustre to the list of brilliant pioneer surgeons. No recovery from oesophageal atresia or tracheo-oesophageal fistula had ever occurred before 1939; now there are several such reported. Swenson (1948) has recorded 57 survivals out of 113 operations. The mortality in congenital diaphragmatic hernia has dropped by 60% in the last fifteen years, and the prognosis in certain types of congenital heart disease and in bronchiectasis has been materially improved. As Ladd (1948) says, the treatment of congenital anomalies may be expected to advance even more rapidly during the next few decades.

Appendicitis—or, rather, peritonitis resulting from delayed diagnosis of acute appendicitis—is still responsible for a formidable number of deaths every year. It occurs at all ages, and unfortunately with the most equivocal signs and symptoms in the youngest infants, when the diagnosis indeed is often not considered. The differential diagnosis of the acute abdomen is a matter calling for clinical acumen of the highest degree, but I am quite sure that in any case of doubt the correct thing is to play for safety by doing a laparotomy.

And what can one say about deaths from road accidents? Though hardly a paediatric problem, it certainly has repercussions that sadden paediatricians as much as everyone else. In 1945, 1,540 school-children were killed in accidents in England and Wales—651 of them on the road. It is a depressing thought, and one that should come home to doctors who are allowed adequate petrol to keep their cars on the road, that one of the advantages of the abolition of the basic petrol ration has been a lowering of the death rate among children from motor accidents.

Conclusion

Research is constantly proceeding all over the world: in the wards of hospitals, in laboratories, and in the field workers of all sorts are engaged on curative and prophylactic problems alike, and many of their puzzles will be solved in due course—doubtless to be replaced by others again in their turn to be solved. As soon as we get an effective bactericidal agent, up crops the resistant strain; as soon as we immunize infants we get a susceptible adult population; as soon as we find the cause of a group of diseases and their specific treatment a new organism

appears—e.g., the virus, still awaiting a virucide. Nature evidently abhors not only a vacuum but stasis also. In paediatrics particularly is it necessary to avoid stasis; for example, the static infant soon becomes marantic, and the marantic infant dies. That is what we have to prevent. There should be no such thing as marantic infants needing cure—they should be so cared for that the causes are prevented. Prophylactic and curative paediatrics must advance together hand in hand if real child health is to be achieved.

REFERENCES

Antonov, A. N. (1947). *J. Pediat.*, **30**, 250.
Cohen, P. (1947). *Med. Clin. N. Amer.*, **31**, 590.
—— and Scadron, S. J. (1943). *J. Amer. med. Ass.*, **121**, 656.
Davison, W. C. (1934). *The Compleat Paediatrician.* Duke Univ. Press, Durham N.C., U.S.A.
Harris, T. N., Grimm, E., Mertens, E., and Ehrich, W. E. (1945). *J. exp. Med.*, **81**, 73.
Jaffe, I. P. (1948). In press.
Kariher, D. H., and Miller, D. I. (1947). *Amer. J. Obstet. Gynec.*, **54**, 1.
Kerpel-Fronius, E., Varga, F., and Kátai Pál (1948). *Arch. Dis. Child.*, **23**, 87.
Ladd, W. E. (1948). *Surg. Gynec. Obstet.*, **86**, 247.
Sako, W., Treuting, W. L., Witt, D. B., and Nichamin, S. J. (1945). *J. Amer. med. Ass.*, **127**, 379.
Swenson, O. (1948). *Pediatrics*, **1**, 195.
Waddell, W. W., and L'Engle, C. S. (1946). *J. Pediat.*, **29**, 487.

TEMPERATURE RECORDING IN SICK CHILDREN

BY

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AND

B. J. HUSSEY, M.R.C.S., L.R.C.P.

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The temperature curve of a sick child is regarded as an essential observation in estimating, for example, the presence or persistence of infection, and a rise above normal in healthy children may be the earliest sign of an infectious malady such as measles or poliomyelitis. This being so, the means of estimating the temperature is obviously of great importance and the definition of "normal" requires careful consideration. Several curious and anomalous temperature curves prompted the small inquiry reported here, which, although it possibly raises more problems than it solves, may serve to stimulate others to larger surveys.

The standard methods of temperature-taking in children in Great Britain involve placing the thermometer in the mouth, the rectum, the axilla, or the groin. It does not seem to be sufficiently realized that considerable differences in the temperature recorded in these four situations may be encountered, and the readings are by no means comparable. For example, in a letter following an article by Green and Penfold (1947) on clinical thermometers as a possible source of cross-infection in hospital, Albury (1947) concludes that "the best plan would be to take temperatures in the axilla rather than in the mouth," as if readings in these two situations were the same. Fig. 1 from the series to be here reported shows a definite pyrexia for mouth readings which would have largely been missed over a period of ten days if the axillary temperature had been accepted against the usual "normal" as shown on a standard temperature chart. Fig. 2 shows a comparison between rectal and axillary readings which equally, in the earlier part, demonstrates a pyrexia by rectal readings which the axillary readings fail to record.

Present Investigation

The investigation consisted of observations on 32 children taken at random in a general medical ward unit. The readings were taken at 6 a.m. and 6 p.m. over a period of six consecutive days, giving twelve readings in each child, from which the differences were calculated. A standard "half-minute" thermometer was used and, unless otherwise stated, was left in the chosen site for three minutes before the

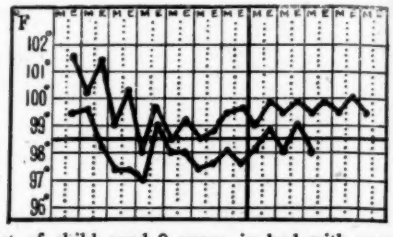

FIG. 1.—Chart of child aged 9 years, in bed with purpura. Upper line = mouth temperature; lower line = axillary temperature.

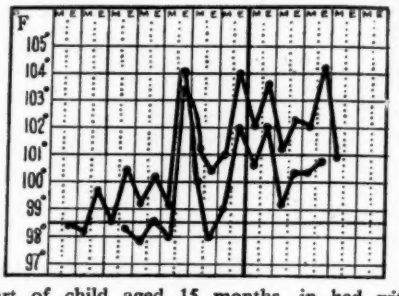

FIG. 2.—Chart of child aged 15 months, in bed with anaemia. Upper line = rectal temperature; lower line = axillary temperature.

temperature was recorded. When two sites are mentioned two thermometers were used simultaneously. The following results were obtained (all figures are the mean of twelve readings):

A. Comparison of groin and rectum

1.	Age 1 month:	rectal reading	1·05° F. (0·58° C.)	higher than groin
2.	„ 2 months:	„ „	0·92° F. (0·51° C.)	„ „
3.	„ 2 years:	„ „	1·16° F. (0·64° C.)	„ „
4.	„ 2 „	„ „	1·18° F. (0·655° C.)	„ „

B. Comparison of groin and mouth

1.	Age 9 years:	mouth reading	0·78° F. (0·43° C.)	higher than groin
2.	„ 7 „	„ „	0·3° F. (0·166° C.)	„ „

C. Comparison of groin and axilla

1.	Age 4 years:	groin reading	0·4° F. (0·22° C.)	higher than axilla
2.	„ 9 „	„ „	0·7° F. (0·39° C.)	„ „
3.	„ 4 months:	„ „	0·4° F. (0·22° C.)	„ „
4.	„ 14 „	axilla „	0·16° F. (0·089° C.)	„ groin

D. Comparison of mouth and rectum

1.	Age 10 years:	rectal reading	0·16° F. (0·089° C.)	higher than mouth
2.	„ 10 „	„ „	0·35° F. (0·194° C.)	„ „
3.	„ 5 „	„ „	0·15° F. (0·083° C.)	„ „
4.	„ 5 „	„ „	0·3° F. (0·166° C.)	„ „

E. Comparison of mouth and axilla

1.	Age 8 years:	mouth reading	1·38° F. (0·766° C.)	higher than axilla
2.	„ 9 „	„ „	0·88° F. (0·49° C.)	„ „
3.	„ 5 „	„ „	0·9° F. (0·5° C.)	„ „

Comparison of rectum and axilla

1.	Age 9 months:	rectal reading	1·08° F. (0·6° C.)	higher than axilla
2.	„ 15 „	„ „	1·6° F. (0·9° C.)	„ „
3.	„ 4 years:	„ „	1·1° F. (0·61° C.)	„ „

Case 1 was running a high swinging temperature, whereas in Cases 2 and 3 the temperatures were between 97° and 100° F.

In the next two groups the temperatures were recorded (with "half-minute" thermometers) after two minutes and after five minutes.

G. Axilla

1.	Age 6 weeks:	5-min. reading	0·75° F. (0·42° C.)	higher than 2-min reading
2.	„ 9 years:	„ „	0·96° F. (0·53° C.)	„ „
3.	„ 5 „	„ „	0·9° F. (0·5° C.)	„ „
4.	„ 6 „	„ „	0·45° F. (0·25° C.)	„ „
5.	„ 10 „	„ „	0·4° F. (0·22° C.	„ „

H. Groin

1.	Age 2 months:	5-min. reading	0·85° F. (0·47° C.)	higher than 2-min reading
2.	„ 2 years:	„ „	1·4° F. (0·78° C.)	„ „
3.	„ 6 „	„ „	0·4° F. (0·22° C.)	„ „
4.	„ 4 „	„ „	0·6° F. (0·33° C.)	„ „
5.	„ 1 week:	„ „	0·5° F. (0·267° C.)	„

Case 2 in this series was that of an abnormally obese child.

Certain other observations were made which serve to confirm what others have recorded, and it would be tedious to give details. For example, the child who is in bed shows a closer correlation between readings in groin and rectum than the child who is up and about the ward. The notorious

"normal" temperature recorded in the out-patient department (usually axillary recording) which became "febrile" when the child was warmed up in bed was seen so often as to be regarded as a routine phenomenon—dangerous as it may be.

Too much must not be deduced from the small number of children observed, but from nearly four hundred recorded temperatures it is justifiable to conclude (1) that the highest temperature recorded is in the rectum, which is approximately 1° F (0.55° C.) higher than in the groin, 0.2° F. (0.11° C.) higher than in the mouth, and 1.3° F. (0.72° C.) higher than in the axilla; (2) that the lowest recording is that in the axilla, which is approximately 0.4° F. (0.22° C.) lower than in the groin and considerably lower than in the mouth and rectum; (3) that the average ("half-minute") clinical thermometer does not attain a maximum reading until at least five minutes when the skin temperature is being recorded in the axilla and in the groin.

No doubt some of these discrepancies could have been avoided with more accurate thermometers and perhaps with more expert observers; but they represent the ordinary everyday methods employed in a hospital ward with ordinary instruments and the observations of competent nurses in training, supervised for the purpose of this investigation by the senior nursing staff.

Discussion

It may be argued that the slight differences discovered are of little significance. It is true that with a case of pneumonia it does not matter greatly whether the temperature recorded is 104° F. (40° C.) rectal or 102° F. (38.9° C.) axillary. But in cases of rheumatic carditis, for example, or primary tuberculosis, a temperature curve never rising above the "normal" marked on the chart as recorded in the axilla or groin may quite mask a low-grade pyrexia revealed by recordings in the mouth or rectum.

These facts have long been known even if in clinical work they are sometimes forgotten. Wright (1945) may be taken as typical of standard teaching. He quotes the normal temperature range (presumably in adults) as 96.7–99° F. (36–37.2° C.) in the mouth and 97.2–99.5° F. (36.2–37.5° C.) in the rectum. He states that the rectal temperature is the highest of the various routes—averaging 0.5° to 0.75° F. (0.267–0.42° C.) higher than the mouth. In warm surroundings (i.e., in bed) he thinks the buccal temperature about equals the rectal. Temperature-recording in the axilla or groin he condemns as "inaccurate in thin subjects" (but note the fat child mentioned above) "and otherwise approximates to buccal." For the purpose of clinical paediatrics children may generally be regarded as "thin subjects," and this condemnation may be viewed in glaring contrast to the usual standing orders to nursing staff in children's hospitals to use the axillary or groin situations. Inquiries made during the present investigation suggest that not all children's physicians are aware of the methods of temperature-recording used for patients under their care. It would appear that the rectal site is best for babies under 1 year and presents little difficulty. For children over 5 years and certainly over 8 the mouth can be used, especially if the tougher type of "rectal" thermometer is employed to avoid the small danger of breakage. Between these ages, in the toddler, the choice is not so easy.

Psychological objections to the rectal site have been urged, but the evidence seems more theoretical than practical. It may be stated in reply that the rectal route at all ages is widely, if not exclusively, used on the continent of Europe, and the incidence of grave psychological trauma has not been seriously advanced for this large section of the world's population. Secondly, as with so many procedures in children, it all depends on the attitude of the nurse dealing with the child. If this is "matter-of-fact" and kindly, and if children are aware of others undergoing the same investigation, it is extremely doubtful if harm is done by the insertion of a thermometer into the rectum. The time factor may be a more serious consideration, for it is generally conceded that the nurse should stand by the child during the rectal reading, whereas skin-recording, and even mouth-recording in older children, can be done for a group at once. What is needed is an instrument which not only claims to be a "half-minute" thermometer but actually records its maximum during this period.

The question of infection is obviously of great theoretical importance, as Green and Penfold (1947) have indicated, an especially when the mouth or rectum is used. The only solution is to have a separate thermometer for each child, kept permanently in a suitable disinfectant between readings. This is, in fact, the practice at many children's hospitals.

For accurate readings the rectal site should be chosen for all children under 5 years and the mouth after this age in most cases. Axillary and groin readings should be given up as inaccurate. Temperature charts should always record which situation has been used.

Summary

Simultaneous temperature-recordings in various situations—mouth, rectum, groin, and axilla—in an unselected series of 32 children show differences of some practical importance. It is argued that the axilla and groin should be discarded as sites for temperature readings. Possible difficulties from the point of view of psychological trauma and infection are discussed.

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REFERENCES

Albury, M. (1947). *Lancet*, **2**, 597.
Green, J. B. M., and Penfold, J. B. (1947). Ibid., **2**, 89.
Wright, S. (1945). *Applied Physiology*, 8th ed., p. 689. London.

INFANTILE DIARRHOEA AND VOMITING

A REVIEW OF 456 INFANTS TREATED IN A HOSPITAL UNIT FOR ENTERITIS

BY

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Units for the treatment of enteritis of infants were established at most of the infectious diseases hospitals administered by the London County Council. The unit at the North-Eastern Hospital came under my care in January, 1945. The cases recorded in this paper were admitted during the years 1945 and 1946. Early in 1947 the unit was moved to more suitable accommodation in single-cell wards. The experience of that year is not comparable with that of previous years and has therefore been excluded.

The object of this paper is to describe the organization and working of the unit, to discuss the type of case treated, and to analyse the results of treatment.

Organization

Accommodation and Equipment.—Throughout the period 1945–6 the unit was contained in two wards of the composite, or Hiorns, type (Harries and Mitman, 1947). Each ward was designed to provide accommodation for 31 patients, distributed as follows: 5 in single cells, 6 in double cells, and 20 in groups of 4 in the five bays of the

main ward. In practice it was found desirable to limit the number of cots in the main ward to 12, with a maximum of 14. The total number of patients in each ward therefore did not exceed 25. Each ward was fully equipped for barrier-nursing. Each bay of the main ward and each cell contained a fixed hand-basin with elbow taps. Two steam sterilizers were available—one for instruments and one for crockery. The ward kitchen contained a refrigerator. In addition each ward was equipped with the instruments and apparatus required for intravenous therapy in infants. A set of sterilized instruments and other necessary apparatus were kept on a trolley, ready for immediate use in urgent cases. Sterile fluids for intravenous administration were supplied from a central laboratory in 500-ml. bottles with screw caps.

Staff.—Both wards were under the direction of the same resident medical officer throughout the two-year period. Off-duty and holiday relief was provided by resident medical officers who had become familiar with the established routine. Uniformity of treatment was thus maintained. Routine medical ward rounds were made three times a day, and the medical officer was available at any time. Each ward was in the charge of a sister who had had general hospital and fever hospital training. So far as was possible, transfers of ward sisters were avoided. The nursing staff comprised fever-trained nurses, general-trained and probationer nurses in training for fever certificates, assistant nurses, and ward orderlies. Frequent changes of nursing staff were unavoidable. The number of nurses allocated to the wards varied widely. At first 11 staff by day and 4 by night were available for each ward of 23 to 25 patients, allowing a ratio of one nurse on duty to every six infants. As the shortage of nurses became more acute the total allocation for each ward (including sister, nurses, and ward orderlies) was reduced to 6 or 7 by day and 2 by night. Consequently, there were generally three, and often only two, nurses on duty in the ward at any one period of the 24 hours. All nurses were familiar with the technique of barrier-nursing and the general management of infants. In the unit they were taught the indications for the institution and termination of intravenous therapy and the methods of maintaining intravenous drip infusions in small infants.

General Management.—Few breast-fed infants were admitted with enteritis. The mothers of these were accommodated with their infants in the cells off the main ward. Maintenance or re-establishment of breast-feeding was usually all that was required for their recovery. The majority of infants with enteritis, and almost all the severe cases, had been artificially fed before admission. For these, feeds were prepared in the kitchen of each ward. All dried-milk feeds were freshly made up as required. Benger's modified milk was prepared twice a day and stored in the refrigerator in the ward kitchen. Hartmann's Ringer-lactate solution (*Extra Pharmacopoeia*, 22nd edition, 1941) was available, sterilized, in 500-ml. bottles.

Prevention of Cross-infection.—The cots were widely spaced in the bays of the main ward, and bed-isolation and barrier-nursing techniques were employed throughout the unit. Infants with parenteral infections associated with diarrhoea and vomiting were, so far as was possible, nursed in complete isolation in the cells off the main ward. The system of "clean" and "dirty" nurses could not always be employed owing to the shortage of staff. Napkin-changing rounds were made two-hourly, and were so arranged as to be completed before the times of feeds. Individual rectal thermometers were supplied for each infant. Separate gowns were worn when attending to each baby. Soiled linen and napkins were placed in closed bins containing "white disinfectant fluid" and sent to the laundry twice daily. No sluicing of napkins or other articles was permitted in the wards. Feeding-bottles, teats and valves, measuring-jugs, and other articles required for preparing feeds were sterilized after each feed and stored in covered containers.

Laboratory Investigations.—The routine investigations were cultures of faeces and rectal swabs for known pathogens, red blood cell counts, and haemoglobin estimations. White cell counts were made in all cases at first, but were found of limited value, and were later confined to selected cases. Other laboratory investigations were made as required, but biochemical investigations other than total-serum-protein estimations were not made. Specimens of fluids for intravenous administration were taken at the beginning and the end of all intravenous infusions and cultured for contaminating organisms.

Treatment

The methods of treatment were based on previously reported experience (Alexander and Eiser, 1944). The underlying principles are of sufficient importance in practice to merit restatement.

1. Careful assessment of the infant's condition on and after admission, with particular reference to (a) clinical evidence of dehydration; (b) its degree and duration; (c) the presence of parenteral infection.

2. (a) Prevention of dehydration by ensuring a sufficient fluid intake; or (b) its prompt correction by the administration of adequate fluids by the appropriate route.

3. A short initial period of starvation followed by an early return to milk-feeding.

4. Adequate treatment of parenteral infections.

The methods employed were made as simple and as uniform as possible consistent with the varying needs of the individual case. The classification of cases with regard to treatment was into two broad groups—namely, those without clinical evidence of dehydration (non-dehydrates), and those with clinical evidence of dehydration (dehydrates). The dehydrates were further subdivided into two groups: (*a*) mild or moderate cases which might be expected to respond to oral rehydration, and (*b*) severe cases for which intravenous therapy was essential. Details of the methods of treatment of artificially fed infants are given below.

Non-dehydrates

An infant who was not dehydrated on admission was given half-strength Hartmann's solution by mouth two-hourly for 18 to 24 hours, in the usual allowance of 2½ oz. per lb. (156 ml. per kg.) body weight per day. No milk feeds were given during this period and no purgatives were permitted.

After 18 to 24 hours dilute milk feeds were given in the form of one-third-strength Benger's modified milk, 2½ oz. per lb. per day. If this was well tolerated the feeds were increased to half-strength Benger's the next day, to two-thirds strength on the day following, and then to three-quarters-strength. The size of the feeds was increased and the intervals between feeds lengthened after the first or second day.

As soon as the stools became normal the feeds were changed to the milk on which the infant had been fed before admission, beginning with a third-strength dilution of the half-cream mixture and working up gradually by daily increases to the full-strength feeds—half-cream in the case of smaller infants and full-cream for infants over 3 months of age or 10 lb. (4.5 kg.) in weight. Those of 6 months and over were given a weaning diet. The infant was discharged as soon as he was able to take an adequate diet with no return of diarrhoea or vomiting and was gaining weight.

If at any time the diarrhoea or vomiting recurred the infant was again given half-strength Hartmann's by mouth for 12 hours and then dilute feeds as before. If, however, diarrhoea was excessive or vomiting was persistent, or if the infant refused feeds or became dehydrated, feeds were stopped and intravenous therapy instituted.

Dehydrates

An infant who was mildly or moderately dehydrated on admission but who was in good general condition and able to take sufficient fluid by mouth was treated on the same lines as those who were not dehydrated. The usual allowance of fluid was given by mouth, plus 5 to 10 oz. (140–280 ml.) per day with the object of restoring the fluid deficit. If dehydration was not readily corrected by this means, or if the general condition deteriorated, oral replacement of fluid was abandoned and intravenous therapy begun without delay.

If on admission dehydration was severe or the infant in a collapsed state intravenous therapy was immediately instituted. These infants were given enough fluid intravenously to satisfy their daily requirements, with a supplementary allowance to replace the fluid lost—usually 10 to 20% of body weight in the more severe cases.

The technique of intravenous fluid administration was in accordance with accepted methods (cf. Garrod, Batten, and Thursfield, 1947). Strict attention was paid to the prevention of local sepsis and contamination of intravenous fluids. Therapy was begun with 300 ml. of half-strength Hartmann's plus 5% glucose given by a slow drip adjusted to deliver the amount required by each infant in the 24 hours. This was followed by 400 ml. of human serum or plasma diluted with 150 ml. of half-strength Hartmann's. For infants weighing less than 7 lb. (3.2 kg.) the initial quantities were halved—i.e., 150 ml. of half-strength Hartmann's plus 5% glucose, followed by 200 ml. of serum diluted with 100 ml. of half-strength Hartmann's.

For the first 18 to 24 hours no feeds were given, but the infant was allowed ½ oz. (14 ml.) of half-strength Hartmann's two-hourly to keep the mouth moist. Small feeds —1 oz. (28 ml.) of half-strength Hartmann's and then of one-third-strength Benger's—were started after this period, the rate of drip being gradually reduced and the amount of feeds slowly increased. The drip was continued, giving alternate Hartmann's and serum, until the infant was fully rehydrated and able to take enough fluid and food by mouth for the daily requirement. If severe diarrhoea, vomiting, or dehydration recurred feeds were stopped and a new intravenous infusion begun. When intravenous therapy was completed the subsequent treatment was as described for non-dehydrates.

If signs of anoxaemia, such as cyanosis or pallor, were present oxygen was administered, using an oxygen tent. Many of the severe cases showed a degree of pallor, and oxygen was freely used on the assumption that this was a sign of incipient circulatory failure. The marked clinical improvement frequently observed would seem to justify this precaution.

Infants with red blood cell counts of less than 2½ millions or haemoglobin readings below 50% were given whole blood intravenously, but only after haemoconcentration had been corrected.

Additional Therapy

If a parenteral infection was found or suspected the infant was treated with one of the sulphonamides. The drug of choice was sulphadiazine given in doses of 0.5 g. four-hourly (3 g. per day) for five days to an infant under 1 year of age. Penicillin, when indicated, was given in doses of 20,000 to 40,000 units intramuscularly at four-hourly intervals for 5 to 7 days. A combination of sulphadiazine and penicillin was thought generally to be more effective than penicillin alone. Sulphadiazine was usually well tolerated and was given orally even to those infants receiving intravenous alimentation. The relatively unabsorbed sulphonamides, such as sulphaguanidine, were not found to be effective in the control of diarrhoea.

The only other drug used was iron, in the form of a ferrous sulphate mixture, for the treatment of some cases of anaemia. Vitamins A, C, and D were given to all infants, and Vitamin K and the B complex when indicated.

General Analysis of Cases

Method of Admission

Administrative procedure had an important bearing on the type of case admitted to an enteritis unit, and must be taken into account before any critical analysis of results is attempted. Admission was arranged centrally by the Public Health Department of the London County Council at the request of medical practitioners. So far as was possible, a patient was sent to the unit nearest to his home, but if no bed was available there he was sent to any unit which had a vacancy. Each unit therefore admitted cases from all parts of the London County Council area.

The criteria for admission accepted by the Public Health Department were: (1) Residence within the County Council area; (2) an age limit of two years; (3) a medical certificate that the case was one of "enteritis" or "gastro-enteritis" and required admission to hospital.

Infants arriving at an infectious diseases hospital in an L.C.C. ambulance and certified as suffering from enteritis had to be admitted. The medical staff could exercise no selection in the matter of admission to the hospital. Admissions therefore included cases of diarrhoea and vomiting of all types and of all degrees of severity. Admission of the case to the enteritis unit, however, was at the discretion of the admitting officer, and infants with diarrhoea incidental to some specific infectious disease could usually be accommodated in other wards of the hospital.

Admissions to Enteritis Unit

During the two-year period 1945–6 the number of infants admitted to the enteritis unit of the North-Eastern Hospital was 686, of whom 541 were under the age of 1 year and 145 were between 1 and 2 years of age. These infants came from all parts of the L.C.C. area. Some were admitted from their homes, some from residential nurseries, and some from general hospitals. The numbers of infants in each of these age groups admitted during each year are shown in Table I. In 53 cases no evidence of disease could be found; 112 infants had other diseases, including bacillary dysentery (36), other specific infections (17), respiratory infections (39), and miscellaneous illnesses (20). All these have been excluded from this series.

TABLE I.—*All Cases Admitted to Unit, 1945–6: Age and Diagnosis*

Year	Age in Years	Total Cases Admitted	Diagnosis		
			No Obvious Disease	Diseases Other than Enteritis	Enteritis
1945 ..	0–1	261	8	24	229
	1–2	97	20	36	41
Total ..	0–2	358	28	60	270
1946 ..	0–1	280	20	33	227
	1–2	48	5	19	24
Total ..	0–2	328	25	52	251
Grand total	0–2	686	53	112	521

During 1946 an effort was made to limit the number of admissions of infants over 1 year of age. Of the 65 infants over 1 year old who had enteritis only two were sufficiently dehydrated to require intravenous therapy and none died. This group will therefore not be considered further in this series. The subsequent analysis deals only with the 456 infants under 1 year of age treated in the unit for diarrhoea and vomiting.

Analysis of Enteritis Cases 0–1 year

Table II gives an analysis of these 456 cases showing the number of cases in each 3-months age group, the number of infants who were dehydrated on or after admission, and the number of deaths. Comparison of the dehydration rates with those quoted in previous series (Campbell and Cunningham, 1941 ; Alexander and Eiser, 1944) shows that

TABLE II.—*Enteritis Cases under 1 year: Dehydration and Mortality*

Age Groups	No. of Enteritis Cases	Dehydrated on Admission	No. Dehydrated after Admission Only	Total Dehydrated on or after Admission	Per cent Dehydrated	No. of Deaths	Fatality Rate % All Cases	Fatality Rate % among Dehydrates
I: 0– 2 months ..	149	54	27	81	54·4	11	7·4	13·6
II: 3– 5 ,, ..	171	84	26	110	64·3	6	3·5	5·5
III: 6– 8 ,, ..	77	41	6	47	61·0	3	3·9	6·4
IV: 9–11 ,, ..	59	16	4	20	33·9	2	3·4	10·0
Total ..	456	195	63	258	56·6	22	4·8	8·5

the cases were as severe as any likely to be encountered in a representative sample of infants with enteritis and comparable with any other such sample.

The majority of the infants (320) were under the age of 6 months on admission, and 17 of the 22 deaths occurred in this group. There were 15 infants under the age of 28 days, and of these two died.

The fatality rates are shown for all cases and for infants who were dehydrated. All the deaths occurred among infants who were dehydrated on or after admission. The usual fall in fatality rate with increasing age is shown, but, owing to the small number of deaths and the small number of cases in age groups III and IV, the differences between age groups do not reach statistical significance in this series.

A total of 258 infants were dehydrated during the course of their illness. Of these, 146 were dehydrated on admission only, 10 (6.8%) of them dying. Forty-nine of the infants dehydrated on admission responded to treatment, but became dehydrated again more than seven days after admission; in this group there were six deaths (12.2%). Sixty-three infants became dehydrated for the first time more than seven days after admission, and of these six died (9.5%). Thus 112 infants relapsed with dehydration, with 12 deaths (10.7%).

TABLE III.—*Cases of Enteritis Treated during each 6-Months Period of 1945–6, with Dehydration and Fatality Rates*

6-Monthly Periods	No. of Enteritis Cases	Dehydration		Deaths		
		No.	%	No.	Fatality Rate % All Cases	Fatality Rate % among Dehydrates
Jan.–June, '45 ..	106	69	65·1	12	11·3	17·0
July-Dec., '45 ..	123	62	50·4	3	2·4	4·8
Jan.–June, '46 ..	85	53	62·4	2	2·4	3·7
July–Dec., '46 ..	142	74	52·1	5	3·5	6·7
Total 1945–6 ..	456	258	56·6	22	4·8	8·5

Of the 236 infants who were dehydrated and recovered 73 were rehydrated by oral administration of fluid. The remaining 163 required intravenous therapy for correction of their dehydration. In all, 244 separate infusions were given.

Table III shows the number of infants treated in the enteritis unit during each of the four six-months periods of 1945 and 1946, the number who were dehydrated and the number of deaths. The number of admissions and the dehydration rates were fairly constant for each year. There was a sharp fall in the case mortality from 11.3% in the first six-months period to 2.4% in the second period, and this latter rate was maintained for the succeeding two periods. The differences between the first and subsequent periods are statistically significant : in each case an exact probability test gives $P < 0.02$.

Parenteral Infections

Among the 456 infants of this series a parenteral infection was found on admission in 238 cases (52.2%). In a further 46 cases a parenteral infection became apparent for the first time after admission. In the remaining 172 cases no evidence of parenteral infection was found throughout

TABLE IV.—*Incidence of Parenteral Infections on Admission and after Admission*

Age Groups I–IV	No. of Enteritis Cases	Parenteral Infections on Admission		Parenteral Infections on and after Admission	Parenteral Infections after Admission Only	Total Parenteral Infections	
		No.	%			No.	%
I: 0– 2 months	149	74	49·7	29	18	92	61·7
II: 3– 5 ,,	171	89	52·0	40	18	107	62·6
III: 6– 8 ,,	77	37	48·0	9	6	43	55·8
IV: 9–11 ,,	59	38	64·4	3	4	42	71·2
Total ..	456	238	52·2	81	46	284	62·3

the stay in hospital. Table IV shows the number of infants in each age group who had parenteral infections on or after admission.

The type of parenteral infection found on admission in each age group is given in Table V. It will be seen that

TABLE V.—*Showing Types of Parenteral Infections Found on Admission in Each Age Group*

Parenteral Infections on Admission	I	II	III	IV	I–IV
	0–2 Months	3–5 Months	6–8 Months	9–11 Months	Total
Upper respiratory infections, excluding O.M.	27	18	13	19	77
Suppurative otitis media alone	7	10	2	1	20
Suppurative otitis media + other conditions	4	10	4	6	24
Bronchitis	8	14	6	6	34
Bronchopneumonia	11	23	7	2	43
Skin infections	8	5	2	1	16
Miscellaneous	9	9	3	3	24
Total parenteral infections on admission	74	89	37	38	238
Total cases of enteritis ..	149	171	77	59	456

the majority were infections of the respiratory tract—the miscellaneous group includes urinary infections, stomatitis, etc.

It is of interest to note that only 44 infants showed evidence of suppurative otitis media on admission. In 32 infants this condition developed during the course of their illness. Thus only 76 of the 456 infants in this series had suppurative otitis media. Nine others had "red drums" associated with upper respiratory infections —these have been classified under the heading of "coryza." In all cases the ears were inspected on admission and whenever a pyrexia or any relapse of the diarrhoea or vomiting occurred. Myringotomies were performed in 49 cases. Mastoidectomy was carried out in only four cases. Post-mortem examinations were made of all but five of the 22 infants who died, and in only one case was pus found in the middle ears at necropsy.

Table VI is an analysis of the association of dehydration with parenteral infection found on admission. Of the total of 456 infants, 238 (52.2%) showed evidence of parenteral infection on admission, and in 218 (47.8%) no parenteral

TABLE VI.—*Relationship of Parenteral Infection to Dehydration on Admission*

Dehydration on Admission	Parenteral Infection + on Admission		No Parenteral Infection on Admission		Total
	No.	%	No.	%	
Dehydration + ..	112	57·4	83	42·6	195
Not dehydrated ..	126	48·3	135	51·7	261
All cases	238	52·2	218	47·8	456

$\chi^2 = 3{\cdot}75$. $P = 0{\cdot}053$.

infection could be found. Among the 195 infants who were dehydrated on admission parenteral infections were found in 112 (57.4%) and were not detected in the remaining 83 (42.6%). A χ^2 test on this Table gives only weak support to any suggestion of association between dehydration and parenteral infection.

Analysis of Deaths

There were 22 deaths in this series. Three infants had overwhelming parenteral infections associated with severe dehydration which failed to respond to intensive treatment. Four infants died of severe parenteral infections which, in retrospect, must be regarded as having been inadequately treated. In one of these the institution of intravenous therapy also appears to have been delayed. In one case bronchopneumonia and an empyema were not discovered until necropsy. No sulphonamides had been administered.

Four cases were moribund on admission. One of these had already been given abundant subcutaneous saline infusions, and was waterlogged and oedematous on arrival. Another of these infants was extremely dehydrated and collapsed. Intravenous therapy was attempted but was unsuccessful, and the child died 15 hours later. Two infants were severely dehydrated on admission, failed to respond to adequate intravenous therapy, and died in five and eight days, respectively. It is thought that in these two cases the dehydration was irreversible.

Two infants, and probably a third, were given amounts of fluid intravenously which proved to be excessive and died of cardiac failure. In one case the routine red blood cell count was inadvertently omitted and a severe anaemia—R.B.C. 1,910,000—was not discovered until the day of death. This infant had a severe bronchopneumonia which failed to respond to chemotherapy. It is possible that she would have recovered with the aid of a blood transfusion.

The four remaining cases failed to respond to apparently adequate treatment of their dehydration.

Discussion

The cases described in this series may be considered a representative sample of infants admitted to an enteritis unit in the London County Council area. They were of average severity, and the clinical findings and results of treatment should therefore be generally applicable to infants admitted to hospital with enteritis.

No discussion of the aetiology of infantile diarrhoea and vomiting will be attempted here. It is of interest, however, that the findings in this series are not in accord with the view that parenteral infections are the main causative factor in the onset of diarrhoea and vomiting. From Table VI it may be seen that in a high proportion of the more severe cases of enteritis parenteral infections were not detected although carefully sought.

Neither can otitis media be implicated as a major factor in the onset or relapse of diarrhoea and vomiting in this series, since it was found in only 76 of the 456 infants. It seems probable that the smallness of the number of mastoidectomies required was due to the more effective methods available for the treatment of suppurative otitis media and to the earlier correction of dehydration.

Parenteral infections, however, when present, may adversely affect the infant's response to treatment of the diarrhoea and vomiting, especially when associated with dehydration. It is essential, therefore, that these should be carefully sought for in all cases and energetically treated. Failure of dehydration to respond to adequate intravenous therapy should suggest the possibility of an associated parenteral infection. In such cases, if there is continued pyrexia with no improvement in the general condition, it has become the custom in this unit to give the infant a full course of sulphadiazine even though no definite parenteral infection has been found. It is considered that this practice is amply justified by cases such as those described in the analysis of deaths.

Dehydration remains the chief cause of the continued high fatality rate of infantile diarrhoea and vomiting, and its prevention or early correction constitutes the most important factor influencing recovery. In this series there were no deaths among the 198 infants who were not dehydrated. Among the 258 infants who were dehydrated there were 22 deaths. With earlier recognition and correction of dehydration nearly half of these deaths might have been prevented.

The ability of the infant to conserve water is relatively less than that of the adult. Therefore in any condition characterized by continued fluid loss dehydration occurs more readily in infants and reaches an irreversible stage more rapidly (Gamble, 1947). The condition which most commonly produces dehydration in infants is diarrhoea and vomiting. It should be remembered that this condition also leads to starvation. Therefore an infant who has become dehydrated is in need of food as well as of water and electrolytes. If these cannot be supplied by the oral route parenteral alimentation becomes essential.

Feeding by the gastric drip method does not permit of complete alimentary rest for the infant with severe diarrhoea and vomiting. Diarrhoea usually continues, absorption is uncertain, and dehydration and starvation are prolonged. This method was tried in a series of 22 dehydrated infants, and dehydration was corrected in only two cases. The remaining 20 infants became more severely dehydrated and required prolonged intravenous therapy. The gastric drip method was therefore abandoned.

Water and electrolytes may be administered by the subcutaneous or intraperitoneal routes, but in all but the mildest degrees of dehydration absorption is unreliable. Neither is it possible to provide the infant with adequate amounts of food by these routes. The intravenous route is the only reliable way of supplying such an infant with water and electrolytes, and food in the form of glucose and serum or plasma.

Early institution of intravenous therapy is essential for infants with diarrhoea and vomiting who are severely dehydrated. By this means complete alimentary rest is assured, dehydration is corrected promptly, and starvation is prevented. It is the practice in this unit to continue intravenous alimentation until diarrhoea has ceased or greatly decreased, dehydration is fully corrected, and the infant able to take sufficient fluid and food by mouth for daily maintenance. If watery diarrhoea or dehydration recur, oral feeding is stopped and intravenous therapy reinstituted.

Approximately 40% of the infants admitted to the unit required intravenous therapy. This could have been administered only in a hospital with the necessary facilities. The success of treatment is therefore dependent on: (1) recognition of dehydration in its early stages and admission to hospital of all but the mildly dehydrated; (2) correct assessment of the degree of dehydration in relation to the requirements of treatment; (3) a team of trained and experienced staff to carry out the necessary treatment. These requirements are best fulfilled by the establishment of self-contained hospital units for the treatment of enteritis.

The recognition of dehydration in its early stages is within the province of all medical practitioners, and the need for early treatment should be more widely appreciated. The assessment of the degree of dehydration and the treatment required in each case is essentially dependent on clinical judgment gained through practical experience. The successful application of these methods of treatment depends upon the training of the medical and nursing staff of the unit.

The figures shown in Table III lend support to this view. The fall in fatality rates after the first six months of 1945 appears to be directly related to the training of the nursing staff, since the technique of treatment was the same throughout the two years and the degree of severity of the cases did not vary from one year to the next. The distribution in age groups did not vary significantly in successive periods, nor during the first six-months period was there any preponderance of the younger age groups, in which a higher fatality rate might be expected. Owing to necessarily frequent changes of staff, in approximately six months most of the nurses in the hospital had worked in the enteritis unit and had become familiar with the routine management and treatment established there. Thereafter, although shortage of staff became a serious problem, all the available nurses were acquainted with the nursing and treatment of the dehydrated infant and were able to maintain the high standard of care necessary for these babies.

In this series 112 infants relapsed with dehydration. Since the nature of the infecting organism or organisms in diarrhoea and vomiting is not yet known, it is not possible to say what proportion of these relapses was due to cross-infection or to re-infection. The possibility of such an occurrence, especially in an open ward, must, however, be borne in mind. The observations in a previous series (Alexander and Eiser, 1944) are of interest in this connexion. In that series the infants were nursed in two different wards—an "open" barrier ward and a single-cell barrier ward. The technique of nursing and treatment were identical, and the numbers of admissions and dehydration rates on admission were the same for both wards. Yet the relapse rate in the open barrier ward was four times as great as that in the single-cell ward.

It seems advisable, therefore, that bed-isolation and barrier nursing should be rigidly enforced in enteritis units, which should, whenever possible, be accommodated in wards of single rooms or cells. By the establishment of such units, staffed by trained medical officers and nurses, much may be done to reduce the number of infants still dying of diarrhoea and vomiting.

Summary

The organization of a hospital unit for enteritis of infants is described and the treatment is outlined.

The results of treatment for the years 1945 and 1946 are detailed: 456 infants under the age of 1 year were treated in the unit during this period; 258 (56.6%) of these infants were dehydrated during the course of their illness. There were 22 deaths, giving a case mortality of 4.8% for all cases, or 8.5% for infants who were dehydrated. There were no deaths among infants who were not dehydrated. Parenteral infections were found on admission in 238 cases (52.2%).

The association between parenteral infections and dehydration is low, and this series does not support the view that parenteral infections are a major factor in the causation of diarrhoea and vomiting. The need for careful search for parenteral infections is, however, stressed from the point of view of treatment.

The importance of prevention or early correction of dehydration, the necessity for intravenous therapy in severe cases, and the need for prevention of cross-infection are emphasized. This implies the provision of adequate facilities and a staff of medical officers and nurses experienced in the technique of barrier nursing and the treatment of dehydration in infants.

It is suggested that these requirements are best fulfilled by the establishment of self-contained hospital units for the treatment of enteritis.

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REFERENCES

Alexander, M. B., and Eiser, Y. (1944). *British Medical Journal*, **2**, 425.

Campbell, R. M., and Cunningham, A. A. (1941). *Arch. Dis. Childh.*, **16**, 211.

Gamble, J. L. (1947). *Chemical Anatomy, Physiology and Pathology of Extracellular Fluid*. Harvard University Press, Cambridge, Mass.

Garrod, A. E., Batten, F. E., and Thursfield, H. (1947). *Diseases of Children*, 4th ed., edited by D. Paterson and A. Moncrieff, Vol. I. Arnold, London.

Harries, E. H. R., and Mitman, M. (1947). *Clinical Practice in Infectious Diseases*. 3rd ed. Livingstone, Edinburgh.

MULTIPLE MYELOMA TREATED WITH STILBAMIDINE AND PENTAMIDINE

BY

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Von Rustizky (1873) was the first to describe the disease of multiple myeloma, the name of plasma-cell myeloma being proposed 27 years later by J. H. Wright (1900). Macintyre (1850) had already described a case of multiple bone tumour, calling it mollities ossium, which had a peculiar "animal matter" in the urine that became evident on gentle heating and disappeared on further heating. He sent the urine to Bence-Jones (1848) for examination, and the substance subsequently became known as Bence-Jones protein. About the same time Dalrymple (1846) gave a clear description of the bony changes in a case he reported on as mollities ossium.

Life insurance tables give the incidence of multiple myeloma as 0.03% of all malignant growths or 3% of all bone sarcomas, but recent collections of cases published suggest that the figures may be higher. Thus Batts (1939) found 40 cases in 200 primary malignant tumours of bone, and Bayrd and Heck (1947) saw 18 in a period of six months. Improved methods of pathological investigation and the attention drawn to the disease by publications have no doubt been largely responsible for the increased number of cases diagnosed.

Multiple myelomatosis is usually described as a fatal disease of middle life, occurring twice as often in males as in females and characterized by multiple tumours growing from the marrow and eroding the bone in a characteristic

manner. It is quite apparent, however, that the condition (see Cases 1, 2, and 6 below) may present as a more diffuse pathological process of the marrow without tumour formation and without the typical areas of rarefaction in the bones (Bayrd and Heck, 1947).

All authorities agree that most cases are not recognized until in a fairly advanced stage of the disease and that the clinical manifestations are very variable and entirely non-specific in origin, although some form of pain as a presenting symptom is fairly constant. Batts found a palpable tumour in 45%, characteristic *x*-ray pictures in 60%, Bence-Jones protein in 50%, and circulating myeloma cells in 3% of 40 cases.

Morissette and Watkins (1942) stress the importance of the blood count as an aid to diagnosis. In 56 cases out of 100 they found myeloma cells in the peripheral blood, and stated that anaemia with eosinophilia and lymphocytosis was frequent. They made particular mention of the apparent "greasiness" of the blood smears, presumably due to the increased serum globulin. Sternal puncture appears to offer the most reliable early information, and increase in the raised serum globulin is valuable corroborative evidence.

Until recently treatment, which consisted mainly of *x*-ray therapy, was entirely palliative, being an attempt to lessen the continual pain. Geschickter and Copeland (1936) state that the average period of survival is two years and that it is uninfluenced by treatment. Ghormly and Pollock (1939) gave the average duration after diagnosis as one year. In 1945 Snapper and his colleagues began treatment with stilbamidine and pentamidine, drugs which had been used successfully since 1939 in the treatment of kala-azar (Adler and others, 1939). The trial of these drugs in the treatment of plasma-cell myeloma was provoked by the fact that in this disease, as in kala-azar, there is an increase in the serum globulin.

Snapper's results were encouraging. In all his cases the pain was favourably influenced. Of 15 patients treated up to January, 1947, 11 could walk on discharge from hospital, although relapses occurred and four have since died. The marked clinical improvement was accompanied by healing of fractures and some recalcification, although an anatomical cure of the disease was not obtained, as myeloma cells could always be found in the marrow.

Method of Treatment

Snapper's (1946a) method was followed, the patients being given a low-protein diet, as animal protein appears to block the effect of the drug. Stilbamidine hydrochloride (May and Baker) was used, made up in 0.15-g. ampoules as a yellow powder. Five millilitres of distilled water and 1 ml. of 2% "proctocaine" were added and the injection was given intramuscularly. The first injection contained 0.05 g. of stilbamidine; the second, containing 0.1 g., was given the following day, and subsequent injections of a full ampoule (0.15 g.) were given daily or every other day. Pentamidine was employed similarly in the terminal phases of two fatal cases (Nos. 4 and 5).

To combat vasomotor reactions 1/100 gr. (0.65 mg.) of atropine was dissolved under the tongue half an hour before the injections were given. Adrenaline, which was kept at hand in case of a severe fall in blood pressure, was never found necessary.

Case 1

A flabby, fat, short, pale-looking man aged 66 was admitted as a hypertensive with marked albuminuria and angina of effort and the probability of a recent coronary thrombosis when the systolic blood pressure fell from 200 to 130. His only complaint apart from anginal pain was of increasing weakness.

Blood examination showed a normocytic normochromic anaemia (Hb 45%) and a leucocyte count of 10,000, with 15% monocytes and a marked neutrophil shift (2% myelocytes, 3% juveniles). Since the cause of the blood changes remained obscure a sternal puncture was done, and this revealed 15% myeloma cells. The red cell series with normoblastic maturation was reduced (12%). The granulocyte series was also reduced (45.5%), and showed some atypical and microcytic forms, with an increase in eosinophils. Lymphocytes (18%) and monocytes (6.5%) were on the high side. Urine examinations, which had previously shown proteinuria with casts, now revealed a considerable quantity of Bence-Jones protein. Serum protein showed an increased globulin (4.29 g.), but the serum phosphatase and calcium were normal. Radiographs of the bones showed no characteristic picture, although a faint patchy rarefaction in the parietal bones could be made out.

A course of stilbamidine was begun, but after a total dosage of 0.45 g. over six days the patient died in uraemia.

Necropsy revealed a hypertrophied and dilated heart, very marked atheroma, and pronounced nephrosclerosis. In the sternum, ribs, and femur the marrow had a mottled appearance with areas of actual haemorrhage set in a background of reddish-pink hyperplastic marrow. There was no macroscopic evidence of bony erosion.

Case 2

A tall, well-built but thin man consulted his doctor about thrombosis in the left calf which followed unaccustomed exercise. He also confessed to increasing weakness and loss of colour over the previous nine months. He developed a fever without any definite signs and complained of some indefinite pains in the right side of the chest. Treatment with sulphadiazine and penicillin was without effect.

On admission to hospital he was found to have a normocytic normochromic anaemia (Hb 44%) and a marked leucopenia (1,000 per c.mm.), the neutrophils showing a pronounced degenerative and toxic shift. Sternal puncture disclosed 24% myeloma cells. The red cell series showing normoblastic maturation was relatively increased (23%); the granulocyte series (17.25%) was greatly reduced and showed numerous toxic and degenerative cells. Lymphocytes (26%) and monocytes (4%) were increased. The urine was normal at all times. The serum globulin was considerably raised (5.6 g.). The serum calcium and serum phosphatase were normal. A radiograph showed only some very indefinite areas of possible rarefaction in the skull.

Transfusions were given to raise the haemoglobin to 76% and a course of stilbamidine was begun. The patient died after 1.15 g. had been given over eleven days. Permission for a necropsy was not obtained.

Case 3

A well-built woman aged 62 complained of vague epigastric and subcostal pain, which was diagnosed as indigestion. A few months later she developed pain and stiffness of the right hip and right shoulder and was treated for rheumatism. A tender firm subcutaneous swelling then appeared over the right clavicle and a radiograph revealed a pathological fracture with the typical appearance of plasma-cell myeloma, such changes also being apparent in the pelvis and both scapulae.

The blood count showed a microcytic hypochromic anaemia (Hb 74%) and a leucopenia (3,200 per c.mm.). Sternal puncture revealed 15% myeloma cells. The red cell series (22%) showed normoblastic maturation with some microcytes; the granulocyte series (36.5%) was reduced, and the lymphocytes (17%) were on the high side. The serum globulin was considerably raised (4.3 g.). The serum calcium (11.8 g.) and the serum phosphatase (41 units) were also raised. The urine was normal.

A total of 4.5 g. of stilbamidine was given over three months with much improvement. The patient was able to get about for short distances and had very considerable relief of pain. There was some recalcification of the clavicular fracture, but the swelling remains unchanged. Radiographs showed no new lesions, although those in the pelvis seemed slightly larger. A dissociated trigeminal anaesthesia appeared six months after treatment started and three months after it ceased. It has now completely resolved.

Case 4

A well-built woman aged 42 consulted her doctor for an aching pain in the middle and lower parts of the back. It had been present for some months. She had recently noticed some breathlessness on exertion and an increasing weakness. Physical examination did not reveal any cause for these symptoms.

The blood count disclosed a macrocytic anaemia (Hb 41%) and a leucopenia (3,800 per c.mm.) due to a reduction in neutrophils, the latter showing a marked shift. There were 3% of myeloma cells in the blood smears, which were particularly "greasy." At the same time a routine radiograph of the chest showed an expanding and bone-destroying tumour of the seventh rib posteriorly. Radiographs of the rest of the skeleton did not reveal any other lesions. A sternal puncture showed 61.5% myeloma cells, with the red cell series (3%) and the neutrophil series (20%) greatly reduced, findings which indicated extensive and widespread involvement of the marrow in spite of the fact that only one small tumour was apparent on the *x*-ray film. The serum globulin (5.6 g.) and alkaline phosphatase (26 units) were considerably raised. The urine was normal.

After a transfusion a course of deep *x*-ray therapy was given without any improvement and the pain became worse. A total of 4 g. of stilbamidine was then given over a period of two months; this resulted in complete alleviation of pain, although the general condition continued to deteriorate and the urine now contained protein and casts. Bence-Jones protein was never found. Over the next two months 3.8 g. of pentamidine was given, but the patient went steadily downhill and died from complete aplasia of the marrow due to replacement by myeloma tissue and renal failure. There were generalized haemorrhages and purpura.

Case 5

A stocky well-built Greek complained in January, 1946, of intermittent epigastric and subcostal pain. He was investigated for peptic ulcer in hospital, but no definite diagnosis was made. Over the next year the pain became much worse, was more extensive and continuous, and was accompanied by increasing weakness.

In June, 1947, the blood count showed an extensive leuco-erythroblastic anaemia (Hb 36%) with 3% myeloma cells in the differential count. A sternal puncture revealed 86% myeloma cells, with very scanty red cell series (less than 1%) and white-cell series (13%). The serum globulin was raised (3.7 g.). The urine contained albumin, casts, and pus, although Bence-Jones protein did not appear until the very terminal stages.

A radiograph of the skeleton taken in June, 1947, showed advanced generalized myelomatosis with considerable collapse of the spine and many fractures. Support was given by a spinal jacket and he received a course of *x*-ray therapy without any improvement or relief of pain.

Stilbamidine therapy was started on Oct. 11 and totalled 8.6 g. over a period of three months, combined with transfusions, folic acid and iron, and penicillin for broncho-pneumonia. Apart from improvement in the pain, the patient gradually became worse and died on Jan. 12, 1948. During the last two weeks 2.1 g. of pentamidine was given, also without improvement.

Necropsy revealed the ribs, sternum, and vertebrae to be almost entirely replaced by myelomatous tissue. In the long bones the marrow cavities were enlarged and filled with such tissue. There were very numerous pathological fractures. Sections and smears showed masses of myeloma cells, about 7% containing inclusion bodies (see below). Both kidneys showed pyelonephritis. Sections of the kidneys and the liver revealed myelomatous infiltration.

Case 6

A large well-built man aged 63 was admitted with generalized purpura, haematuria, epistaxis, melaena, and a right testicular haematoma. Generalized aching pains had been present for the last few months, had become worse in the last few weeks, and were now accompanied by some loss of feeling in the hands and feet. Marked arteriosclerosis and some chronic bronchitis were present. The central nervous system, apart from rather weak jerks, showed no abnormality.

Blood examination showed a normal red cell count, a thrombocytopenia, a leucocyte count of 10,000 per c.mm., with a marked neutrophil shift and an eosinophilia (23%), and the presence of scanty myeloma cells. A sternal puncture revealed 9% myeloma cells with 4% plasma cells. The red cell series (14%) and the neutrophil series (31%) were reduced; eosinophils (18%) were increased. The serum globulin (7.8 g.) was greatly raised. The serum calcium (11.8 mg.) and plasma alkaline phosphatase (16.9 units) were slightly increased. The urine contained protein with some granular casts, but no Bence-Jones protein. A radiograph of the skeleton failed to show any abnormalities. A total of 4.2 g. of stilbamidine was given over two months. Clinical improvement was at first limited to disappearance of the purpura, but on completion of the treatment the patient could get about fairly well and said that his pains were not as bad.

Effects of Treatment

Clinical.—Case 3 has shown definite improvement with relief of pain and could get about for short distances twelve months after the diagnosis was made and approximately two years from the time of the first symptom. Case 6 has shown some improvement and is fairly mobile six months after the diagnosis and twelve months from the time of the first symptom. In Cases 4 and 5 the patients, who had very extensive marrow involvement before treatment, died after a period of some months without showing any improvement except for the relief of pain. In Cases 1 and 2 the patients died suddenly after a few injections of stilbamidine, the first from renal failure and the second from agranulocytosis.

On Myeloma Cells.—Snapper and his colleagues demonstrated the development of basophilic inclusion bodies in the cytoplasm of myeloma cells in patients receiving stilbamidine. These bodies appeared to be compounds of stilbamidine and ribose nucleic acid. It is suggested that the ribose nucleic acid becomes dissociated from its protein in the ribose nucleic complex and attached to the stilbamidine to form a substance which appears within the cytoplasm of the cell as deeply basophilic granules. These granules ultimately coalesce to form inclusion bodies. Such inclusion bodies were demonstrated in Cases 3, 4, and 6 (Figs. 1 and 3). Case 5 never developed them in spite of the large dosage and the length of treatment. Cases 1 and 2 ended fatally before there was time for them to develop. A reduction in the number of myeloma cells was obtained by sternal puncture in Case 3 (15% to 3%) and in Case 6 (9% to 5.5%) (Table I). In both cases the small number of myeloma cells originally present in the peripheral blood disappeared.

FIG. 1.—Two typical myeloma cells, one containing at least 12 well-formed inclusion bodies and the other three earlier and less distinct bodies. (×850.)

Biochemical.—No improvements were obtained except in Cases 3 and 6 (Table II). In Case 3 the plasma globulin, the alkaline phosphatase, and the serum calcium returned to normal. In Case 6 they showed a fall towards normal.

Haematological.—Cases 3, 4, and 6 developed a very considerable eosinophilia in the early stages of treatment (74% in Case 6). In Case 4 this persisted until death, but in

TABLE I.—*Myelograms*

Case No.	Myeloma Cells			Plasma Cells	Reticulum Cells	Haematocytoblast	Red Cell Series				White Cell Series									
												Neutrophils					Eosins			
	Primitive	Typical	Multinuclear				Pro-erythroblast	Early Normoblast	Intermediate	Late Normoblast	Blast Cells	Pre-myelocyte	Myelocyte	Juvenile	Band	Segment	Early Eosin.	Eosin.	Lymphocytes	Monocytes
Normal mean values:	0·5	14	0·5	3	1	1	1	1	8	3	0·5	1	7	9	8	14	3	3	18	6·5
1	2	20	2	6	0·5	0·5	3	3	7	10	0·75	2	4	1·5	2	4	2·5	1·5	26	4
2	2·5	11	1·5	6·5	0·5	1	1	3	11	7	0·5	1	6·5	6	5·5	14·5	1	1·5	17	2·5
3 (*a*)	V.S.	2	1	3	0·5	1	1	7	16	9	2	2·5	12	10	7	9	1	1	13	2
(*b*)	2·5	50	9	4	1	V.S.	V.S.	V.S.	1	2	V.S.	0·5	2	2	6	8	1	1	10	1
4	3	82	1	2	0·5	V.S.	V.S.	V.S.	V.S.	V.S.	V.S.	0·5	2·5	2	2	1·75	V.S.	V.S.	2·5	0·25
5	—	9	Occ.	4	0·5	0·5	0·5	1	5	7·5	0·5	2	12	8	4	5	9	9	8	1
6 (*a*)	0·5	5	—	4	0·5	0·5	0·5	2	4·5	11	0·5	1·5	18	8	3	11	6	10	14	2
(*b*)																				

(*a*) = before treatment. (*b*) = after treatment. V.S. = Very scanty

TABLE II.—*Biochemical Findings*

Case No.	Serum Proteins (G. per 100 ml.)					Blood Urea (mg./100 ml.)	Plasma Alk. Phos. (Units)	Serum Calcium (mg./100 ml.)
	Total	Alb.	Glob.	Glob. as % Total	A : G Ratio			
1	9·14	4·9	4·24	46	1·15 : 1	60 rising to 270	10·6	10·5
2	9·38	3·77	5·61	60	0·7 : 1	40	9·7	10·7
3 (*a*)	7·7	3·4	4·3	56	0·7 : 1	38	41	11·8
(*b*)	7·25	5·4	1·85	26	4 : 1	36	9	9
4	8	2·4	5·6	70	0·43 : 1		24	10·4
5	7·3	3·6	3·7	51	1 : 1	35 rising to 100	—	—
6 (*a*)	11	3·2	7·8	71	0·4 : 1	45	16·7	11·8
(*b*)	10·2	3·5	6·7	65	0·5 : 1	35	14	11

TABLE III.—*Blood Counts*

Case No.	Hb%	R.B.C.	C.I.	M.C.V. (c.μ.)	Remarks	Leucocytes	Neutrophils				Eosins.	Lymphos.	Monocytes	Myeloma cells	Remarks
							Segment	Band	Juvenile	Myelocyte					
1	45	2·5	0·9	89	Size, shape, and staining normal	10,000	36	17	3	2	3	24	15	—	Blood film greasiness with rouleaux formation +
2	44	2·3	0·96	91	,, ,,	1,000	24	22	—	—	2	43	9	—	Greasiness with rouleaux formation ++
3 (*a*)	74	4·9	0·77	78	Microcytic and hypochromic	3,200	25	11	2	1	2	48	11	2	Greasiness with rouleaux +. Developed eosinophilia in treatment
(*b*)	81	4·7	0·86	88	Size, shape, and staining normal	3,600	37	10	—	1	9	36	6	—	No greasiness or rouleaux formation
4	41	1·9	1·1	105	Macrocytic and normochromic variation +	3,800	21·5	23	2	0·5	1	42	7	3	Greasiness with rouleaux ++++. Developed 36% eosinophils in treatment
5	36	2·2	0·8	89	Variation in size, shape, staining +++	4,400	29	22	3	1	5	32	5	3	Greasiness with rouleaux ++++. Developed 20% eosinophils in treatment
6 (*a*)	90	5·0	0·9	90	Size, shape, and staining normal	10,000	46	7	2	1	23	18	3	Scanty	Greasiness with rouleaux +. Developed 74% eosinophils in treatment
(*b*)	90	5·2	0·8	86	,, ,,	7,000	53	9	—	—	8	22	8	—	Thrombocytopenia improved from 80 to 120,000

(*a*) = before treatment. (*b*) = after treatment.

Cases 3 and 6 fell towards normal at the end of treatment and has remained at approximately 8% since. No improvements in the general blood picture were obtained except in Cases 3 and 6 (Table III).

Toxic.—These were not marked. All cases except two complained of nausea following the injection, and actual vomiting occurred in Cases 1, 5, and 6. A dissociated trigeminal anaesthesia occurred in Case 5, but was followed by complete recovery.

Local.—Complaints of pain at the site of injection varied but did not appear to be a distressing factor except in Case 6, in which in spite of the use of a local analgesic the injections came to be anticipated with dread.

Description of the Abnormal (Myeloma or Plasma) Cells Found in the Marrow

The origin of the cells found in multiple myeloma has been greatly disputed, and descriptions given have incriminated most of the early cells found in marrow and in particular myelocytes, erythroblasts, lymphoblasts, and reticulo-endothelial cells.

The myeloma cells found in these cases were divided into three types—blast cells (Fig. 5), typical myeloma cells (Figs. 2 and 3), and multinuclear cells (Fig. 4)—and the following descriptions are given with the suggestion that the typical myeloma cell resembles a cell intermediate between a blast type (plasma blast) and an adult plasma cell and compares in cellular age and position with the myelocyte.

The typical myeloma cell was oval in shape and about 16 μ in its largest diameter, and had a round nucleus situated towards one pole and occupying half the cell space. The nucleus was confined by a definite nuclear membrane and the distinction between its parachromatin and chromatin was marked, the latter appearing as stippling or as larger aggregations arranged irregularly but not infrequently leaving a pale central zone which gave the

impression of a dispersing nucleolus. The cytoplasm stained irregularly, giving a foamy appearance. As a rule deeply basophilic, it had a definite and distinctive purplish tint (Jenner-Giemsa stain), often contained a number of azure granules, and was sometimes vacuolated.

The primitive or blast type (Fig. 5) was about 26 μ in diameter. A definite nuclear membrane enclosed a nucleus which occupied three-quarters of the cell space and possessed one large nucleolus with sometimes up to three small nucleoli in addition. The chromatin assumed a definite stippled pattern. The cytoplasm presented the same features as described in the typical myeloma cell but did not contain granules or vacuoles nor stain so deeply.

FIG. 2.—Early stage in development of typical myeloma cell: disappearance of nucleoli and condensation of nuclear chromatin. Outline of the foamy cytoplasm distorted by spreading. (×965.)

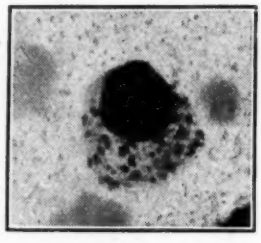

FIG. 3.—Typical myeloma cell containing stilbamidine inclusion bodies. Pale central nuclear zone surrounded by coarse chromatin masses. (×965.)

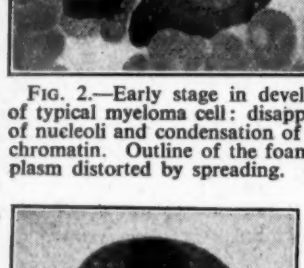

FIG. 4.—Multinucleated myeloma cell. (×965.)

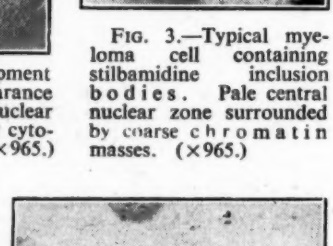

FIG. 5.—Blast type of myeloma cell containing three nucleoli. (×850.)

The multinuclear cells usually contained two nuclei but occasionally had four or more, and varied in size from 20 to 40 μ. The nuclear and cytoplasmic characteristics were identical with those described in the typical myeloma cell.

In all cases the myelogram showed an increase in the number of normal plasma cells—i.e., plasma cells indistinguishable morphologically from those found in normal marrow.

Comments

The fact that stilbamidine has such a favourable influence on the pain of multiple myeloma constitutes an advance in the treatment of this malignant disease. The method of administration by intramuscular injection is simple and not usually accompanied by severe local or general reactions provided the precautions outlined are taken. In addition stilbamidine seems to have a specific effect upon the protoplasm of some myeloma cells which is evident in fixed stained preparations. While this effect does not bring about complete disappearance of the cells there is evidence that it hinders their increase and dissemination, allowing local healing reactions to take place. The fact that no other cells but myeloma cells are affected suggests that the protoplasm and protein metabolism of the latter differs from that of other cells. Snapper (1946a) states quite definitely that stilbamidine has no effect in other malignant diseases such as leukaemias and Hodgkin's disease.

Two prime factors seem to influence the success of treatment: (1) the initial blood count findings, which are a reflection of the state of the marrow, and (2) the extent of renal involvement.

Those cases which presented with a serious anaemia did not improve. The malignant process was evidently too widespread and had replaced too much normal marrow to permit recovery of haemopoiesis. It seems likely that not enough stilbamidine could be given to have an effect on all the myeloma cells. For example, in Case 5, which received 8.6 g., inclusion granules were demonstrated in only 7% of the myeloma cells, and this figure did not vary significantly whether the tissue examined was from the ribs, sternum, vertebrae, or femur.

The one patient showing extensive renal involvement (Case 1) died in uraemia after only six injections, suggesting that the stilbamidine had provoked an unfavourable renal reaction.

Snapper (1946a) stated that stilbamidine gave its best results in patients with widespread but not large osteolytic lesions and with normally functioning kidneys. Detailed information on the blood counts and marrow findings was not given.

Summary

Reference is made to some of the literature on multiple myeloma, the difficulty of early diagnosis, and the help that pathological investigation may give.

A detailed description is given of six cases and their treatment with stilbamidine by Snapper's method. (Two cases also had pentamidine). Four of the cases were fatal and two have made some clinical recovery. These results do not compare very favourably with those of Snapper, who reported seven out of fifteen showing continued improvement.

The outstanding clinical feature of treatment with stilbamidine is the relief from pain.

The morphological features of the abnormal cells found in the marrow and the appearance of inclusion bodies in their cytoplasm as the result of treatment are described.

My thanks are due to Dr. H. V. Morlock, Dr. Harold Davis, Dr. Lindsey Batten, Dr. K. Blatchley, Dr. Gray Thomson, and Dr. Z. Leitner for the opportunity of studying these cases, to the laboratory and nursing staffs for their co-operation, and to Mr. J. H. Hunter for the photomicrographs.

REFERENCES

Adler, S., and others (1939). *Ann. trop. med. Parasit.*, **33**, 313, 327.
Batts, M. (1939). *Arch. Surg.*, **39**, 807.
Bayrd, E. D., and Heck, F. J. (1947). *J. Amer. med. Ass.*, **133**, 147.
Bence-Jones, H. (1848). *Philos. Trans.*, p. 55.
Dalrymple (1846). *Dublin J. med. Sci.*, **2**, 85.
Geschickter, C. E., and Copeland, M. M. (1936). *Tumors of Bone.* New York.
Ghormly, R. K., and Pollock, G. A. (1939). *Surg. Gynec. Obstet.*, **69**, 648.
—— —— Hall, L. B., and Beizer, L. H. (1942). Ibid., **74**, 242.
Macintyre, W. (1850). *Med.-chir. Trans. Lond.*, **33**, 211.
Morissette, L., and Watkins, C. H. (1942). *Proc. Mayo Clin.*, **17**, 433.
Snapper, I. (1946a). *J. Mt Sinai Hosp.*, **13**, 119.
—— (1946b). *Blood*, **1**, 534.
—— (1947). Ibid., **2**, 311.
—— *et al.* (1947). *J. Amer. med. Ass.*, **133**, 157.
Von Rustizky, J. (1873). *Dtsch. Z. Chir.*, **3**, 162.
Wright, J. H. (1900). *Johns Hopk. Hosp. Rep.*, **9**, 359.

The Executive Board of WHO has authorized the Director-General to sign a preliminary agreement, as soon as 14 of the American Republics have deposited their instruments of ratification of the WHO constitution, between WHO and the Pan American Sanitary Organization providing for the ultimate establishment of that organization as the WHO regional organization for the western hemisphere. The Executive Board has also recommended that WHO establish contacts with the following international non-governmental organizations: International Union against Venereal Diseases, International Union against Tuberculosis, International Union against Cancer, World Federation for Mental Health, International Committee of the Red Cross, International Hospital Federation, International Academy of Forensic and Social Medicine, International Leprosy Association, International Association for the Prevention of Blindness.

SEPSIS IN RELATION TO TUMOURS

CLINICAL IMPLICATIONS OF AN EXPERIMENTAL STUDY

BY

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The occasional occurrence of sepsis in surgical wounds maintained under rigid aseptic conditions leads to the inference that such infection can take place by routes other than direct contact from without. Learmonth (1924) described invasion of undamaged muscle of an arm by *Clostridium welchii* following a partial gastrectomy. Okell and Elliott (1935) demonstrated the transient presence in the blood of organisms associated with oral sepsis. Buchanan and Kyles (see C. H. Browning, 1944) by repeated blood cultures showed the occurrence of bacteriaemia after minor surgical intervention in a limited area where infection is present, as, for example, in the extraction of teeth. Such circulating organisms are no doubt usually disposed of rapidly by the blood, as indicated by the work of Wright and Douglas (1903), but where persistent bacteriaemia exists and there are sites of lowered tissue resistance conditions will be favourable for the establishment of secondary metastatic infection.

Butler and Valentine (1943) stressed the importance of small initial staphylococcal lesions in maintaining bacteriaemia—e.g., those occurring under hard skin or beneath a scar. Damage following slight trauma will supply conditions favourable for secondary localization, and degenerating tumour tissue would also appear to afford an excellent nidus in which pyogenic organisms may settle. Muir (1941) has stated that tumours have little resistance to bacterial invasion, and Boyd (1947) emphasized the part played by sepsis as a cause of death in cancer of the human subject.

Experimental Investigation

It was therefore decided to ascertain experimentally whether such abnormal tissues are in fact especially apt to harbour pyogenic organisms brought by the blood stream. The problem resolved itself into balancing the factors of tumour growth and progress of infection so that the effects of both might be assessed. A rat sarcoma originally induced by styryl-430 (P. Browning, 1941) provided a readily transplantable tumour with a regular progressive course of sufficient duration to permit observation both of the effect of the new growth and of the bacterial agent.

Following a subcutaneous injection of a coarse emulsion of about 0.05 g. of tumour tissue in saline, death takes place in six to eight weeks from pulmonary metastases, or the animal requires to be chloroformed owing to ulceration over the grafted tumour. The strain of staphylococcus was that used by Browning and Calver (1947), as it responds well to treatment with penicillin. It is noteworthy that rodents are naturally resistant to staphylococcal infection, apparently owing to their plasma being little affected by the coagulase of the organism (Hale and Smith, 1945); however, virulence may be enhanced in various ways—e.g., by addition of mucin to the inoculum. In order to reduce so far as possible the effects of toxin production *in vitro* the inoculum for a rat of approximately 200 g. consisted of 1 ml. of a 1 in 20 dilution of an eighteen-hour agar growth injected intraperitoneally.

It was necessary to control the infection by chemotherapy, three or four doses each of 1,000–2,000 units of penicillin being injected subcutaneously in the first twenty-four hours after inoculation. This treatment was the minimum necessary to effect cure in the majority of inoculated control animals without tumours, 36 out of 43 being cured. Those which did not respond to the therapy died from septicaemia within a few days. In the survivors there were no focal lesions apart from occasional transitory suppuration in the needle-track of inoculation which appeared about a week after infection and which lasted for only a few days. Out of 52 inoculated but untreated animals 43 died within twenty-four hours from staphylococcal septicaemia; within seven days the nine survivors had to be chloroformed owing to extensive sepsis located in the inoculation track.

Tumour-bearing rats were inoculated seven to twelve days after grafting, when the growths were about 1 cm. long. Out of 62 which were inoculated and treated with penicillin as above described 52 survived; these were chloroformed before growth of the tumour led to surface ulceration (about five weeks after inoculation). Immediately after death approximately 0.1 g. of the tumour was cultured in nutrient broth; several loopfuls of spleen tissue and heart blood were also cultured and other organs examined for lesions, and it was noted that no septic focus existed at the site of inoculation. From the tumours in 19 out of the 52 animals coagulase-positive staphylococci were recovered (often in mixed culture with coliform organisms and diphtheroids). On the other hand, the tumours of all 21 uninoculated tumour-bearing control animals, which had received no penicillin, yielded no staphylococci, although coliform organisms and diphtheroids were often present. Staphylococci were not demonstrated in the spleen or heart blood of either the treated tumour series or the controls. In some of the tumours the staphylococcus was isolated where no gross central degeneration had occurred. These experiments show that localization of pyogenic organisms in a malignant tumour tends to occur as a result of bacteriaemia.

Conclusions

Two conclusions of clinical importance follow from the results. First, especially in view of Butler and Valentine's work, minor sepsis should be regarded seriously in cancer patients, particularly when radiation therapy may lead to marked lowering of the local resistance. Secondly, since it has been shown that the virulence of pyogenic and other organisms is greatly enhanced by the injection of adrenaline at the site of inoculation (Evans, Miles, and Niven, 1948), where surgery is associated with the use of combined local analgesic and adrenaline the prior clearing up of septic foci which might otherwise seem of little consequence is of prime importance.

I acknowledge with thanks a grant towards the expenses of this work from the British Empire Cancer Campaign.

REFERENCES

Boyd, W. (1947). *A Textbook of Pathology*, p. 259, London.
Browning, C. H. (1944). *Glasg. med. J.*, **141**, 1.
Browning, P. (1941). British Empire Cancer Campaign Annual Report, p. 184.
—— and Calver, K. M. (1947). *J. Path. Bact.*, **59**, 417.
Butler, E. C. B., and Valentine, F. C. O. (1943). *Lancet*, **1**, 194.
Evans, D. G., Miles, A. A., and Niven, J. S. F. (1948). *Brit. J. exp. Path.*, **29**, 20.
Hale, J. H., and Smith, W. (1945). Ibid., **26**, 209.
Learmonth, J. R. (1924). *Lancet*, **2**, 648.
Muir, R. (1941). *A Textbook of Pathology*, p. 206, London.
Okell, C. C., and Elliott, S. D. (1935). *Lancet*, **2**, 869.
Wright, A. E., and Douglas, S. R. (1903). *Proc. roy. Soc.* **B, 72**, 357.

The American National Multiple Sclerosis Society, which was formed two years ago to stimulate interest and finance research in disseminated sclerosis, has appointed as medical director Dr. Cornelius H. Traeger. The appointment was made possible by a grant of $50,000 from the Milbank Memorial Fund. Dr. Traeger is a member of the Executive Committee of the New York Rheumatism Association and of the Committee of Research and Education of the American Arthritis and Rheumatism Foundation.

ANTIHISTAMINE DRUGS AND RADIATION SICKNESS

BY

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AND

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Radiation sickness is a syndrome characterized in varying degree by drowsiness, fatigue, nausea, and vomiting. Indirect evidence in favour of the hypothesis that histamine plays a part in the production of this syndrome has been put forward by various workers (Segal, 1939 ; Leblond and Segal, 1942 ; Ellinger, 1945, 1946, 1948 ; Thygesen, 1947). The use of antihistamine drugs in the treatment of radiation sickness has been advocated by Lofstrom and Nurnberger (1946). These authors claimed that the administration of "benadryl" to patients who had developed radiation sickness resulted in a considerable amelioration of symptoms. However, in their paper not enough details were given of the factors governing *x*-ray dosage and there was no control series of cases. In view of this claim, and also of the possible significance of histamine in the production of radiation sickness, it was considered that a controlled clinical experiment was required to clarify the position.

Details of the Experiment

The antihistamine used was "anthisan," which has been shown to be the most potent and specific drug available (Schild, 1947 ; Gaddum, 1948). The patients used for the investigation were those undergoing post-operative irradiation with *x* rays for carcinoma of the breast. This form of treatment has been standardized in respect of duration of treatment, volume irradiated, and total dosage given (McWhirter, 1948). The 47 patients in this study were divided into three groups, as follows:

Group A.—Seventeen patients were given anthisan in doses of 0.6–1 g. daily. Ten of these received 1 g. daily and seven had 0.6 g. daily, these latter cases being unable to tolerate the higher dose. The active tablets were administered for five days before starting irradiation so that side-effects were minimized and an adequate concentration of anthisan was secured in the tissues.

Group B.—Fifteen patients were given an inactive preparation identical in appearance to the active preparation. This was also administered for five days before the beginning of irradiation.

Group C.—Fifteen patients were given no preparation at all.

In every case an assessment of the patient's clinical condition was made by one of us (W. M. C. B.) who was entirely ignorant of the type of tablet which the patient was being given. This assessment was made after every fifth treatment in respect of drowsiness, fatigue, headache, nausea, and vomiting. The results are shown in the Table:

Group	Total No. of Cases	No. Developing Radiation Sickness
A	17	16 (94·1%)
B	15	10 (66·6%)
C	15	11 (73·6%)

It will be noted that only a small number of patients have been studied in each group. The investigation had to be abandoned because many of the patients given the active drug became so upset after the starting of irradiation that they would have been unable to continue with *x*-ray treatment unless the drug had been withdrawn.

Toxic Effects.—Five out of the 17 cases receiving anthisan developed a toxic erythema accompanied by pronounced constitutional symptoms during irradiation. This patchy erythema, first noted in the posterior shoulder field between the third and fifth days after the beginning of *x*-ray therapy, spread centrifugally, becoming generalized over the greater part of the trunk and extremities after 48–72 hours. Withdrawal of the antihistamine drug at any stage in its development resulted in a rapid disappearance of the erythema even though *x*-ray therapy was continued. Such a skin rash never occurs with *x*-ray therapy alone, and is seen in less than 1% of cases receiving antihistamine drugs for other purposes.

Discussion

The important practical point that emerges from the study is that in spite of the small number of patients observed it can confidently be stated that anthisan is of no value in the treatment of radiation sickness. Any benefit which results from the administration of other antihistamine drugs is likely to be due to properties other than their antihistamine effect. In addition, not only did anthisan fail to prevent radiation sickness, but, as will be seen from the Table, it appears that those patients receiving the active drug showed a greater incidence of constitutional upset while having *x*-ray therapy than did either of the two control groups. The difference between Group A and the control series is not statistically significant, but was convincing enough to stop the trial of further cases.

There is considerable evidence to show that histamine may be released in animals in a variety of circumstances. The similarity which exists between the manifestations of certain human states and conditions that can be produced experimentally in animals has led to the belief that histamine plays an important part in certain disease processes. Dale (1948) has suggested that it may possibly have a physiological role in the control of capillary tone. Though there is no direct proof that histamine is released from the tissues by ionizing radiations, the presumption of such a release is an attractive hypothesis which explains many of the manifestations of radiation sickness.

This study seems to show that the administration of anthisan during irradiation with *x* rays produces a toxic erythema in some cases and may also give rise to a profound constitutional upset. Antihistamine drugs such as anthisan exert their effects by modifying the tissues in such a way that locally released histamine does not produce its pharmacological effects. It is suggested that the local release of histamine resulting from irradiation with *x* rays and leading to vasodilatation and alteration in capillary permeability may be of physiological importance, and that this type of reaction to injury may well play an important part in preserving the integrity of the organism. The administration of agents such as antihistamine drugs during irradiation may interfere with this protective mechanism, thus accounting for the marked effects noted in this study.

We are indebted to Professors McWhirter, Dunlop, and Gaddum for their advice and criticism in the construction of the experiment and during the preparation of the paper. Anthisan tablets and dummy tablets were supplied by Messrs. May and Baker, Ltd.

REFERENCES

Dale, H. (1948). *British Medical Journal*, **2**, 281.
Ellinger, F. (1945). *Radiology*, **44**, 241.
—— (1946). *Science*, **104**, 502.
—— (1948). *Radiology*, **50**, 234.
Gaddum, J. H. (1948). *British Medical Journal*, **1**, 867.
Leblond, C. P., and Segal, G. (1942). *Amer. J. Roentgenol.*, **47**, 302.
Lofstrom, J. E., and Nurnberger, C. E. (1946). Ibid., **56**, 211.
McWhirter, R. (1948). *Proc. R. Soc. Med.*, **41**, 122.
Schild, H. O. (1947). *Brit. J. Pharmacol.*, **2**, 189.
Segal, G. (1939). *C. R. Soc. Biol., Paris*, **131**, 1079.
Thygesen, J. E. (1947). *Acta radiol., Stockh.*, **28**, 1.

PATENCY OF VEINS AFTER A NEW TECHNIQUE OF CUTTING-DOWN FOR TRANSFUSION

BY

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AND

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During the course of a research project which necessitated the introduction of a catheter into a vein of an arm we felt somewhat guilty at depriving each patient of a vein which might be required later in an emergency that would conceivably need every available transfusion site. As our patients were all women, and also pregnant, this matter seemed very important. A technique was devised whereby the patency of the vein could be retained for any future occasion. This extremely simple method can be used for any type of transfusion where the introduction of a cannula is necessary. We recommend it particularly to junior house officers, who occasionally waste veins once and for all time. With this method they may still waste a vein through their inexperience, but only temporarily.

Method

The vein is exposed and mobilized in the usual way, using a small transverse incision, and a loop of fine catgut is drawn underneath by means of an aneurysm needle (Fig. 1).

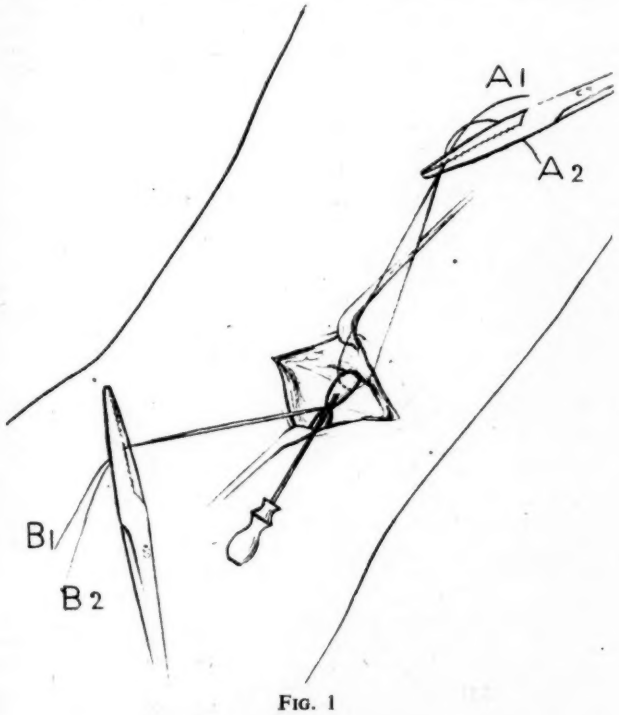

FIG. 1

This loop is then cut so that there are two pieces of catgut —the upper piece, A_1 and A_2, and the lower piece, B_1 and B_2. The upper piece is not tied, but its two ends are secured with a pair of Spencer Wells forceps.

The lower piece is tied once only and the ends secured. A small nick is then made in the vein wall between the two pieces of catgut and the cannula is inserted. It will then be necessary to tie A_1 and A_2 together to secure the cannula, but this step is not necessary in catheter work.

At the end of the transfusion or research catheterization the loop A_1 and A_2 is untied and the free ends are brought through the upper skin edge. Similarly with B_1 and B_2, the free ends being brought through the lower skin edge (Fig. 2).

A_1 is then tied to B_1 and traction applied to the free ends A_2 and B_2. A few drops of penicillin are applied locally, the cannula withdrawn, and A_2 and B_2 tied together. The wound is then closed, and bleeding from the vein is controlled by direct pressure against the skin. It will be seen that there is no buried catgut, and the loops—A_1 and A_2, B_1 and B_2—come out spontaneously after five to seven days. Few cases of infection occur, and what infection does arise is very mild and is easily treated along usual surgical lines. As a prophylactic measure we recommend the introduction of 200,000 units of penicillin along the catheter as it is withdrawn. The final scar is minute.

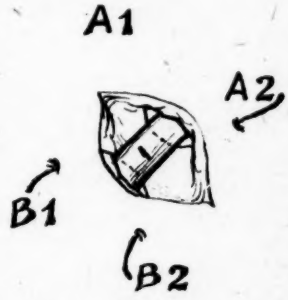

FIG. 2

A refinement of the technique which makes this operation simpler, affords better control of the vein, and minimizes the chances of infection is to bring the cut ends of each loop out on the skin before nicking the vein wall.

We have been able to show that the veins thereafter remain patent in 100% of cases not only by inserting a needle but also by cutting down on them again in the course of our research.

Medical Memoranda

A Note on Direct Inguinal Hernia

Although commonly associated with advancing years, the direct variety of inguinal hernia is not infrequently found in younger men who are not necessarily of poor physique, and it is occasionally seen under the age of 20. For some of the older subjects surgical treatment cannot be entertained; but to others, especially those in active occupations, operation would be more acceptable than a truss if the results were less uncertain. Methods of repair are very diverse, and recurrence rates vary widely between figures of 4.4% (McCloskey and Lehman, 1940) and 35% (Page, 1943).

The narrow strip of conjoint tendon or the free margin of the internal oblique muscle which bounds the broad defect cannot be brought down to the inner end of Poupart's ligament. A Bassini repair is therefore unsuitable and carries an average recurrence rate of 17.6% (Mair, 1945). Instead it is necessary to fill in the intervening gap with fascia or other material which will remain in place permanently. As this gap lies directly underneath the external ring and the weak intercolumnar fascia, it appears advisable also to make full use of the external oblique by displacing the exit of the spermatic cord as far outwards as possible. Subcutaneous transplantation of the cord is not harmful (Rose and Carless, 1940), but doubt has been expressed about the value of the external oblique aponeurosis (Gallie and Le Mesurier, 1924). Yet at operation for recurrent hernia the aponeurosis is found to be an intact sheet, tough and fibrous, the sac emerging from an aperture left for the passage of the cord. Overlapping is less important than closure right down to the pubis.

In Service cases particularly the possibility must be kept in mind that operation may be marred by side-effects, especially upon the locomotor system, for which reason the routine use of fascia cut from the thigh is open to question. As an alternative I sought to apply the older method of McArthur (1901), for the deep half of the repair, to a consecutive unselected series of 55 direct herniae in 38 such patients. Most of them were aged 30 to 40, with extremes of 20 and 45. Five herniae were

of the curious and uncommon funicular type described in Maingot's *Post-Graduate Surgery* (1937), the rest diffuse; five were recurrent, of which one was funicular.

Generally the broad-based sac was not cut away and the transversalis fascia not considered strong enough to be worth any attention. Disturbance of the internal ring was limited to the removal of seven small oblique sacs. The first couple of turns of the fascial stitch picked up the rectus sheath and were drawn fairly taut so as to bring the rectus sheath and Poupart's ligament as close together as possible. This closed the inner part of the defect, narrowed the remainder, and left enough of the suture to lace the internal oblique loosely to Poupart's ligament. All areolar tissue under the external oblique and upon the pubis was cleared away and resuture of the aponeurosis started on the pubis. A satisfactory strip could not be obtained from either the upper or the lower leaf of the external oblique in only one instance—a recurrence with an ugly appendicectomy scar close by and much fibrotic matting of the tissues; in this case a Halsted repair was effective.

Results were sought by questionary between two and nearly six years after operation (average interval four years). Twenty-nine replies were received relative to 42 fascial repairs, including the four relapsed cases. One-third of the replies were sent by doctors. There were three recurrences. In one, originally bilateral, an associated small oblique sac reappeared on the outer side of the displaced cord. After I excised this the groins withstood without further trouble three years' artillery service followed by eighteen months' heavy road work. The only traced simple funicular hernia recurred. A third case was reported to have a definite bulge on the iliac fossa on coughing and an impulse felt through the external ring also. Recurrence was simulated in a fourth case by a venous impulse in the superficially placed cords on coughing, but on exploration the repairs proved to be sound. With the possible exception of one patient who was invalided for other causes and later died, all the others have led lives of normal activity, though seven have experienced minor symptoms. Thus, one man has a feeling of weakness on one side, another wears a truss "for extra support," and five have some discomfort at times, probably due to intermittent compression of the cord veins in their abnormal course through the musculature.

The series closely resembles that of McCloskey and Lehman's 45 direct herniae with two recurrences, both in results and in details of repair. I have dealt more recently with fifteen more herniae of this kind, including three that were recurrent, at ages ranging from 18 to 54. The same procedure was found to be practicable in all of them, but the later results are not yet known.

It is reasonable to conclude that the method described is very generally applicable to cases of direct inguinal hernia and satisfactory in a fair proportion. Unless the full technique of Gallie and Le Mesurier (1924) is carried out there is not much advantage in using the fascia lata; but a single suture from any source is unlikely to be of much value unless supported by close contact with the overlying aponeurosis.

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REFERENCES

Gallie, W. E., and Le Mesurier, A. B. (1924). *Brit. J. Surg.*, **12**, 315.
McArthur, L. L. (1901). *J. Amer. med. Ass.*, **37**, 1162.
McCloskey, J. F., and Lehman, J. A. (1940). *Ann. Surg.*, **111**, 610.
Mair, G. B. (1945). *British Medical Journal*, **2**, 178.
Page, C. M. (1943). *Proc. R. Soc. Med.*, **38**, 185.
Rose, W., and Carless, A. (1940). *Manual of Surgery*, 16th ed., Baillière, London.

The production of two new scientific sound films is announced by Crookes Laboratories, London. They are entitled "Emulsions" and "The Story of Halibut Oil," and may be borrowed free of charge by schools, colleges, hospitals, and scientific societies. "Emulsions," which runs for fifteen minutes, opens with a series of simple test-tube experiments showing the contrast between an emulsion and a suspension. The two types of emulsions and the function of emulsifying agents are then demonstrated. The preparation of emulsions experimentally and industrially by mechanical, chemical, and supersonic means and their uses in daily life are described pictorially. "The Story of Halibut Oil" is a thirty-minute film divided into two parts, the first giving vivid details of life aboard a trawler in Icelandic waters, the second concerned with the scientific treatment of halibut liver, formerly a waste product, for the extraction of its vitamin-bearing oil. Both films are available in 16-mm. and 35-mm. sizes and can be supplied at fourteen days' notice.

Reviews

ESSENCE OF OBSTETRICS

Management in Obstetrics. By Andrew M. Claye, M.D., F.R.C.S., F.R.C.O.G., Oxford Medical Publications. (Pp. 186; illustrated. 12s. 6d.) London: Geoffrey Cumberlege (Oxford University Press). 1948.

This is an unusually attractive book on the management of pregnancy and labour, both normal and abnormal. The author does not discuss the aetiology and diagnosis of various conditions except when they determine a particular treatment. It is a very practical book, and with a few minor exceptions, attributable no doubt to the interval between writing and publication, is thoroughly up to date. There is no room for views other than the author's, and these, clearly based on personal and extensive experience, he presents concisely and dogmatically. In obstetrics there is often more than one good way of doing things, so, as Professor Claye points out, it is inevitable that not everyone will accept some of the opinions expressed. Practising obstetricians will largely agree with his views, however, and even those who disagree most can hardly fail to respect and enjoy this reasonable and honest account of how to manage the everyday problems of midwifery.

Each of the 36 chapters—essays is perhaps a more fitting term—is on a single subject or form of treatment; none is long, and some occupy less than a page. Each, headed by a humorously apt quotation, is a joy in itself, not only for its subject matter but for the style and charm of its presentation. Even the details of antenatal care—which, despite its importance, is one of the most tedious subjects to read about—become interesting when Professor Claye discusses them. The book is not intended to describe the management of all obstetrical conditions; much of its attractiveness would be lost if it did. A notable omission is any account of eclampsia. In general, the author has made a happy choice of subjects which cover the essence of modern obstetric practice, and that without the "ifs and buts" which sometimes tend to obscure the picture in the standard textbooks. He is not concerned with the technical details of caesarean section but he is concerned with when and how to carry out episiotomy, how to repair a perineal tear, and how to avoid or treat post-partum haemorrhage.

This handy little book (a coat pocket will easily accommodate it, and three or four hours suffice to read it), full of good sense, is evidently intended primarily for the general-practitioner obstetrician, but all with a genuine interest in obstetrics, and irrespective of their eminence in this branch of medicine, will find in it a fascination which is the attribute of anything well done.

T. N. A. JEFFCOATE.

FATIGUE

Fatigue and Impairment in Man. By S. Howard Bartley, Ph.D., and Eloise Chute, M.A. Foreword by A. C. Ivy, Ph.D., M.D. (Pp. 429. $5.50.) New York and London: McGraw-Hill Book Company. 1947.

The industrial and social disturbances of the recent years have aroused increasing interest in the practical and academic aspects of "fatigue." No excuse need be offered for the publication of any intelligent contribution, nor indeed, since our progress in understanding this problem has been so sluggish, for any novelty of approach. Herein lies the value of this book, whose authors realize that fatigue "has been used more often as an explanation than as something to be explained" and have made a clear and insistent distinction between "fatigue," a subjective phenomenon describable only in personalistic terms, and "impairment," which refers to specific tissue changes identifiable only through physiological and biochemical studies. Fatigue itself they regard "as an experiential pattern arising in a conflict situation in which the general alignment of the individual may be described as aversion," and the limpness, tiredness, feeling of inadequacy for further activity, and sense of bodily discomfort, besides being attempts at retreat or escape, are to be recognized as being fatigue and not merely symptoms of it.

In most of the twenty chapters of this book the authors review material selected from sources discussing the psycho-

logical, physiological, and industrial aspects of problems relating directly to fatigue or to certain conditions which are most likely to induce or influence it. Thus we find discussions on various opinions about fatigue, mental fatigue, chronic fatigue, industrial experiences, conflict, and frustration; and on such topics as continuous activity, anoxia, lack of sugar, temperature extremes, salt deficiency, drug actions, sleep, and dietary habits. The authors pay special attention to neuromuscular activity and aspects of visual functioning. Indeed, it was while examining visual activities that S. H. Bartley formulated his concept of fatigue and was led to test it further. His background of specialized training in psychology and biology, and his researches in neurophysiology and vision, have influenced its elaboration considerably. He is at present professor of research in the visual sciences at the Dartmouth Eye Institute, and Eloise Chute, a psychologist, is his research associate.

The book is not easily read. Many of its chapters give the impression of a conducted tour of physiology, normal and under certain stresses, with concluding comments that impairment of tissues is obvious but that there is nothing which really relates to fatigue. The main virtue of the book lies in the authors' dogged insistence on the origin and nature of fatigue, which is not to be described, detected, measured, or ascribed to any condition in terms of tissue impairment, from which it must be clearly distinguished.

H. M. WHYTE.

PARAVERTEBRAL BLOCK

Paravertebral Block in Diagnosis, Prognosis, and Therapy. Minor Sympathetic Surgery. By Felix Mandl, M.D., F.I.C.S. Translated by Gertrude Kallner, M.D. With foreword by Max Thorek, M.D., F.I.C.S. (Pp. 330. 32s.) London: William Heinemann Medical Books. 1947.

After a short but adequate account of the regional anatomy the author describes fully the various techniques of paravertebral injection. But the book is much more than a mere account of technique. He discusses the application of sympathetic block in many diseases, sets down all the indications for it, and with praiseworthy candour mentions those lesions in which this treatment has proved of no avail. To report failures, or rather non-successes, is helpful and worthy of being copied. The section on angina is excellent, and in the chapters on disorders of the peripheral circulation he finds a place for accounts of many rare and little-known conditions. The book appeals to one as the outcome of much personal experience and wise reading set out with judgment and great fairness.

Inevitably the work here and there lags behind modern practice—the printed book cannot keep pace with the research worker in so live a subject. Phenol, for example, is mentioned at some length, but the author states that it has not been used in practice. There are, however, to-day many patients in whom paravertebral phenol block has been used with complete success, and the question how far this, or a similar method, may replace open operation is part of the research of the immediate future. There is no mention of the use of radio-opaque materials with the injection: the information given by them is often helpful and sometimes chastening. This book is an important contribution to the surgery of the autonomic system. As it contains many facts not easy to come by, it should appeal to all research workers in this field.

E. D. TELFORD.

Diseases of the Chest (E. and S. Livingstone; 25s.), by Dr. Robert Coope, is already becoming recognized as a classic of its kind, and the second edition will be welcomed. For the student there is no better introduction to chest disease and he should not be deterred by its length. The author's style and human approach to his subject make reading easy and enjoyable. But this is not only a book for students; the practitioner will find it a useful guide to treatment, and indeed all who are interested in chest disease will read it with pleasure and profit. Most specialists are at times in danger of losing touch with reality, and it is refreshing to be brought back to the things that really matter. "The clinician," says Dr. Coope, "devotes himself by training and tradition to caring for the one sheep that is lost," and this epitomizes the object of his book. In this edition the author has thoroughly revised the introductory section on anatomy, but otherwise there has been little change. The next edition might well include a somewhat fuller account of modern physiotherapy in chest disease.

BOOKS RECEIVED

[Review is not precluded by notice here of books recently received]

L'Infection de Foyer. By I. Goia. 2nd ed. (Pp. 191. 300 francs.) Paris: Librairie Maloine. 1948.

A monograph on focal infection.

Klinische Hämatologie. By Hanns Fleischhacker. (Pp. 624. Sch. 190.) Vienna: Wilhelm Maudrich. 1948.

An illustrated textbook of haematology.

Les Mathématiques de L'Hérédité. By G. Malecot. (Pp. 63. 180 francs.) Paris: Masson. 1948.

The mathematics of probability applied to genetic inheritance.

Therapy Through Interview. By S. G. Law, M.D. (Pp. 313. 27s.) London: McGraw-Hill. 1948.

An introduction to psychotherapy intended specially for the general practitioner.

Eclampsie et Eclampsisme. By H. Vignes. (Pp. 217. 450 francs.) Paris: Masson. 1948.

A study of eclampsia and pre-eclampsia.

Nursing for the Future. By E. L. Brown, Ph.D. (Pp. 198. $2.00.) New York: Russell Sage Foundation. 1948.

A study for the future education of nurses in the U.S.A.

Pregnancy Diagnosis Tests. By A. T. Cowie, B.Sc., M.R.C.V.S., Ph.D. (Pp. 283. 15s.) Edinburgh: Commonwealth Bureaux of Agriculture. 1948.

A review of the literature, particularly with reference to domestic animals.

La Gelure. By M. S. Vagliano. (Pp. 255. 650 francs.) Paris: Masson. 1948.

The symptomatology and treatment of frost-bite

Introduction to Diseases of the Chest. By J. Maxwell, M.D., F.R.C.P. 3rd ed. (Pp. 307. 12s. 6d.) London: Hodder and Stoughton. 1948.

The author emphasizes the necessity for *x*-ray examination and includes new material on the sulphonamides and antibiotics.

The Dark Mind. By R. Goyne. (Pp. 256. 9s. 6d.) London: Stanley Paul. 1948.

A tale of crime and scandal.

Sex and Citizenship. By E. F. Griffith, M.R.C.S., L.R.C.P. 4th ed. (Pp. 224. 8s. 6d.) London: Methuen. 1948.

The author discusses the relation between religion and sex, and the significance of personal behaviour on the problems of social morality.

Medical Research in France During the War 1939-45. By J. Hamburger. (Pp. 306. No price.) Paris: Flammarion. 1948

Includes papers on the prophylaxis of diphtheria and typhus, antihistamine substances, intravenous procaine, and the treatment of endometriomas with male hormone. In English.

Le Enervazioni Articolari. By F. Morin and F. Roasenda (Pp. 138. 1,400 lire.) Turin: Minerva. 1948.

An account of innervation of the joints for the surgeon.

The 1948 Year Book of Radiology. Edited by Jenner Hodges, M.D., and I. I. Kaplan, M.D., F.A.C.R. (Pp. 472. 36s.) London: H. K. Lewis. 1948.

Abstracts from recent papers on diagnosis and therapy, with editorial comment.

Die Reaktion der Pupille auf Mydriatica nach Unterbrechung der Sympathischen Pupillenbahn. By P. Wormser. (Pp. 160. 17 Swiss francs.) Basle: S. Karger. 1948.

An analysis of pupillary reactions to midriatics after interruption of the sympathetic innervation.

The Countryman Book. Compiled by J. W. Robertson Scott, C.H. (Pp. 308. 10s. 6d.) London: Odhams Press. 1948.

A selection of articles of rural and horticultural interest from the *Countryman*.

Traitement des Dermatoses Communes. By A. Desaux. (Pp. 1,300. 1,960 francs.) Paris: Masson. 1948.

A textbook intended for the general practitioner or young specialist.

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THE KING'S HEALTH

The medical profession in Britain has shared to the full the universal alarm felt about the state of the King's health. His medical advisers in the bulletin issued on Nov. 23 stated that "the King is suffering from an obstruction to the circulation through the arteries of the legs, which has only recently become acute; the defective blood supply to the right foot causes anxiety." The King's doctors underlined the gravity of the news in the bulletin when they stated that "it would be hazardous for his Majesty to embark upon a long journey, which might delay his recovery and which might well involve serious risk to a limb." On Monday of this week a further bulletin brought better news of an improvement in the general health of the King, "an important factor in re-establishing arterial circulation to the feet, which is also being encouraged by appropriate medicinal and physical measures." The King's doctors state that there is less cause for immediate anxiety regarding the right foot.

The medical profession will join with the rest of the community in heartfelt sympathy with the King in his present disablement—a sympathy heightened by their knowledge of the pathology of arterial disease. The British Medical Association in particular will wish its Royal Patron a speedy return to better health.

VIRUSES OF HEPATITIS

There is now no doubt that a certain number of persons in apparent health carry with them in their blood stream a virus which when inoculated parenterally into other healthy human beings will produce in a proportion of cases the signs and symptoms of hepatitis. Evidence that such a virus existed first appeared in 1934 shortly after the introduction of immunization against yellow fever, when the vaccine was made up in pooled serum obtained from presumably healthy persons.[1] Shortly afterwards Soper and Smith,[2] in South America, also reported the occurrence of hepatitis after the administration of yellow fever vaccine, and McNalty[3] drew attention to the fact that the disease had been observed in children and adults who had received inoculations of pooled measles immune serum. Thus even before the war the possibility was recognized that serum from apparently healthy persons might contain an icterogenic agent.[4 5] Unfortunately neglect of this possibility led to much illness in the American armed Forces,[6] while among British Service men and women inoculated in this country nearly 800 cases are known to have occurred.[7] Since human serum has been discarded in the preparation of yellow fever vaccine some hundreds of thousands of persons have been inoculated without the development of a single case of hepatitis.

Meantime there were other pointers to the presence of a virus in human blood and its products. Serviev and his colleagues[8] in Russia noticed the development of jaundice in persons inoculated with a vaccine against sandfly fever; the vaccine contained human serum. Beeson[9] in 1943 described hepatitis appearing one to four months after the transfusion of blood or plasma. The disease has also been known to follow the giving of blood or its products as a prophylactic against mumps[10] and when therapeutic malaria has been induced by the injection of red cells infected by malaria parasites.[11 12] Further presumptive evidence that a virus was present in the blood stream had been derived from the experience of venereal disease clinics in the years between the two world wars. The common belief, however, was that the jaundice was due to arsenic acting on a liver damaged by syphilis, despite the fact that hepatitis was known to occur in patients who had received only bismuth, in patients given injections of acriflavine,[13] and in diabetic clinics.[14] In a survey of the information available on the incidence of arsphenamine jaundice in 1939 Findlay[15] reached the conclusion that some factor other than these drugs was necessary for its development, and that this factor was a virus and quite possibly the virus of infective hepatitis.

The suggestion that a virus was responsible for the production of "syringe-transmitted hepatitis," as it was called, was again made in 1943 by MacCallum[16] and Bigger.[17] Later MacCallum[18] showed that serum from a patient with so-called arsenical jaundice would produce hepatitis in normal volunteers who were not receiving arsenic or any other drug. Since then it has become evident that if carefully sterilized syringes are not used for each individual there is a risk of transmitting hepatitis by giving intramuscular injections of penicillin or even by simple venepuncture. The presence of a hepatitis-producing virus in the blood stream has now been demonstrated throughout Europe, Russia, North and South America, and in various parts of Africa, including Egypt, Uganda, and West Africa.[19]

While these are known facts, there is still room for speculation. Is this virus which is present in the blood stream of perhaps 5%, or it may be even 10%, of apparently healthy persons the same as the virus of infective hepatitis? Though the signs and symptoms of the hepatitis following yellow fever vaccination were indistinguishable from those of infective hepatitis the incubation period was

[1] Findlay, G. M., and MacCallum, F. O., *Trans. R. Soc. trop. Med. Hyg.* 1937, **31**, 297.
[2] *Amer. J. trop. Med.*, 1938, **18**, 111.
[3] *Annual Report of the Chief Medical Officer of the Ministry of Health*, 1937–1938. H.M.S.O., London.
[4] Findlay, G. M., MacCallum, F. O., and Murgatroyd, F., *Trans. R. Soc. trop. Med. Hyg.*, 1939, **32**, 575.
[5] Findlay, G. M., *J.R. Army med. Cps*, 1940, **74**, 72.
[6] Turner, R. H., Snavely, J. R., Grossman, E. B., Buchannan, R. N., and Foster, S. O., *Ann. intern. Med.*, 1944, **20**, 193.
[7] Findlay, G. M., Martin, N. H., and Mitchell, J. D., *Lancet*, 1944, **2**, 301, 340, 365.
[8] *Terap. Arkh.*, **18**, 595.
[9] *J. Amer. med. Ass.*, 1943, **121**, 1332.
[10] *Lancet*, 1943, **1**, 83.
[11] Chalmers, T. C., *J. clin. Invest.*, 1947, **26**, 1055.
[12] Smith, M. H., and Hall, J. W., *J. Lab. clin. Med.*, 1948, **33**, 998.
[13] Murray, D. H., *J.R. Army med. Cps*, 1930, **54**, 19.
[14] Graham, G., *Lancet*, 1938, **2**, 1.
[15] *Recent Advances in Chemotherapy*, 1939, 2nd ed. London: J. and A. Churchill.
[16] *Brit. J. vener. Dis.*, 1946, **22**, 151.
[17] *Lancet*, 1943, **1**, 457.
[18] Ibid., 1945, **1**, 342.
[19] Findlay, G. M., *Mon. Bull. Min. Hlth*, 1948, **7**, 2 and 32.

much longer—58 to 134 days, as against 15 to 35 days for infective hepatitis.[14] Further, the disease tended not to infect contacts. The histological changes in the liver were indistinguishable,[20 21] and neither virus can be transmitted to laboratory animals. However, there have now been outbreaks such as those recorded by Smith and Hall[12] and Capps and his colleagues[22] in which the incubation period was in every case quite short (17 to 43 days); other similar outbreaks are known. Aycock and Oren[23] suggest that the prolonged incubation period of syringe-transmitted hepatitis is due to the presence of immune body together with virus in the inoculum. Although syringe-transmitted hepatitis is not as a rule highly infectious there are instances where contact cases of jaundice are very difficult to explain except by spread of infection.[24 7] Some outbreaks of infective hepatitis in schools, however, have been limited to one or two cases. More recently it has been found that infective hepatitis virus is present in the stools, and in the urine also when haematuria occurs. The virus which is present in the blood stream in syringe-transmitted hepatitis does not appear to be present in the stools, though it conceivably might be if the patient suffered from haemorrhage from the bowels or even from bleeding piles. So far no instance has been published of infection with the blood-borne virus by the alimentary route, whereas infective hepatitis can be readily transmitted orally. Cross-immunity experiments are contradictory: in some immunity to the two viruses appears to develop, in others no immunity. But it must be remembered that the immunity to infective hepatitis is by no means absolute, and cases of a second attack are not uncommon.[7]

This uncertainty whether two separate viruses or strains of one and the same virus are concerned is reflected in the question of nomenclature. There is no doubt about the nomenclature of infective hepatitis: this term was first used by Cockayne[25] in 1912 and thus has priority over "infectious" or "epidemic," which was first used by Lindstedt[26] in 1919. The term "homologous serum jaundice" was used for the disease caused by the blood-borne virus in 1943 in a memorandum prepared by medical officers of the Ministry of Health.[10] Since then a fuller knowledge of the disease has been obtained. The word "jaundice" is unfortunate, for clinical jaundice may be either slight or absent. The fundamental lesion is a hepatitis, so that "homologous serum hepatitis" or, as it has been called in America, "S.H. disease" would be preferable. But the word "serum" is open to criticism, for the infection is transmitted often by plasma and sometimes by whole blood as well as by serum. Findlay[19] suggested the provisional term "haematic," which was preferred to "haematogenous" because the latter suggests that the virus is actually produced in the haemopoietic system.

If the natural method of transmission of haematic hepatitis was known with certainty it might be possible to take more reasonable steps to prevent it. It does not seem to be transmissible by food, but there is evidence that it can infect through the nasopharynx.[27 28] At present we have no means either of preventing natural infection with haematic hepatitis or of determining who is a symptomless carrier of the virus. It is known that after an infection some viruses, such as that of anaemia of horses, remain in the blood stream for life. Whether this is true of the haematic viruses is not yet known. The prevention of haematic hepatitis has two aspects: (1) prevention of cross infection from the use of contaminated syringes and needles; and (2) avoidance of infected blood or its products in transfusions or therapeutic immunizations. In clinics and wards where repeated injections have to be given, often with a limited number of syringes, the only way of avoiding haematic hepatitis is always to clean and boil syringes and needles before each injection. If these steps are correctly carried out there will be no cases of hepatitis. That it is possible to make mistakes even with apparently foolproof techniques is clearly shown in the papers which we published last week by Malmros and his colleagues (p. 936) in Sweden and by Morton (p. 938) in this country. It is only when all concerned in the sterilization of syringes and needles fully realize the necessity for scrupulous care that it will be possible to abolish syringe-transmitted hepatitis.

The selection of uninfected blood, plasma, or serum for transfusion or immunization is a more difficult matter, since there is no indication of who is or is not a carrier of a hepatitis virus. The danger of hepatitis has not always been fully appreciated by those who collect blood from volunteers. Thus in some instances blood has been taken in areas where an outbreak of infective hepatitis was occurring, while persons who have had hepatitis have acted as donors, having been told that if the disease occurred two years previously there was no danger. For this statement there is of course no scientific evidence, since it is not known whether after an attack virus may not at times recur in the blood. By limiting pools of whole blood or plasma to 10 donors it is possible to some extent to limit the distribution of haematic hepatitis virus if the incidence of infected donors is below 1 in 10.

Since the virus may apparently be inactivated by ultraviolet light Wolf and his co-workers[29] gave irradiated plasma to 21 volunteers without untoward reaction, but they offered no evidence to show that the virus was actually inactivated. MacCallum[30] failed to inactivate the virus with the doses of ultra-violet light recommended by Oliphant and Hollaender,[31] but Blanchard and his colleagues[32] have now shown that a practical and effective method of irradiating icterogenic serum and plasma can be evolved. Irradiation cannot of course be applied to whole blood. Gellis and his co-workers[33] used heat in an attempt to inactivate hepatitis virus. Certain viruses (Theiler's mouse encephalomyelitis virus, vaccinia, tobacco-mosaic, and tobacco-necrosis virus) added to pooled plasma

[20] Roholm, K., and Iversen, P., *Acta path. microbiol. scand.*, 1939, **16**, 427.
[21] Dible, J. H., McMichael, J., and Sherlock, S. P. V., *Lancet*, 1943, **2**, 402.
[22] *J. Amer. med. Ass.*, 1948, **136**, 819.
[23] *Amer. J. med. Sci.*, 1947, **214**, 483.
[24] Propert, S. A., *British Medical Journal*, 1938, **2**, 677.
[25] *Quart. J. Med.*, 1912, **6**, 1.
[26] *Nord. med. Ark.*, 1919, **51**, 583.
[27] Findlay, G. M., and Martin, N. H., *Lancet*, 1943, **1**, 678.
[28] MacCallum, F. O., and Baver, D. J., ibid., 1944, **1**, 622.
[29] *J. Amer. med. Ass.*, 1947, **135**, 476.
[30] *Proc. R. Soc. Med.*, 1946, **39**, 655.
[31] *Publ. Hlth Rep., Wash.*, 1946, **61**, 598.
[32] *J. Amer. med. Ass.*, 1948, **138**, 341.
[33] *J. clin. Invest.*, 1948, **27**, 239.
[34] Berk, J. E., *Gastroenterology*, 1947, **8**, 296.
[35] Duncan, G. G., *Amer. J. med. Sci.*, 1947, **213**, 53.
[36] Stokes, J., Jr., Blanchard, M., Neefe, J. R., Gellis, S. S., and Wade, G. R., *J. Amer. med Ass.*, 1948, **138**, 336.
[37] *Gastroenterology*, 1947, **9**, 28.
[38] *J. Amer. med. Ass.*, 1947, **135**, 268.

survived fractionation and were found in all fractions prepared from plasma. Albumin solutions to which the haematic hepatitis virus was added were heated at 60° C. for 10 hours and when inoculated into volunteers failed to cause hepatitis, whereas the unheated mixture of albumin and virus caused hepatitis, though without jaundice, in volunteers. This method of heat inactivation may be applied to albumin solutions, since they can be stabilized to withstand heating; it is not of course applicable to serum, plasma, or whole blood.

Initial studies suggested that gamma globulin injections might be of value in preventing the onset of haematic hepatitis, but more recent investigations[34-36] showed that one injection was valueless. Since there is evidence that passive immunity resulting from the injection of gamma globulin does not last for more than three weeks and the incubation period of haematic hepatitis may be as long as three months, a series of injections would probably be necessary. Further, the large-scale use of gamma globulin would be difficult in a civilian population. It is obvious that much more work requires to be done on the hepatitis viruses. Rosenthal[37] found that 4.08% and Brightman and Korns[38] 4.5% of transfused patients developed hepatitis. More blood and blood products are now being used for transfusion than during the war, and the need for increased knowledge is urgent.

TREATMENT OF INFANTILE ENTERITIS

The announcement early this week that a gastro-enteritis "flying squad" is now based on the Hospital for Sick Children, Great Ormond Street, and ready to work, has roused the interest of the public in this dangerous disease. Deaths from gastro-enteritis in infants under one year of age have exceeded 3,000 per annum since 1940, giving mortality rates which have varied from 4.4 to 5.2 per thousand live births. The increase in the number of deaths in the past few years (3,611 in 1946) is doubtless related to the increased number of births and to the overcrowded and unhealthy houses in which many babies are being reared, for infantile enteritis is a social disease with its greatest incidence among artificially fed infants living in poor-class urban areas. Unfortunately, because this infection is not generally notifiable, our knowledge of its epidemiology is limited, although the investigation by the Wrights[1] was a brave attempt to disentangle some of the environmental factors concerned in its aetiology. Meanwhile the search goes on in the laboratory for a specific causal organism. Here again progress is hampered because the material admitted to hospital may consist of a variety of clinical syndromes varying from simple dietetic upsets, through enteritis associated with some parenteral infection, to primary gastro-enteritis. It is therefore more profitable to concentrate laboratory investigations on localized institutional outbreaks or on more widely spread epidemics. In such an epidemic which occurred in Aberdeen in the spring of 1947 the labours of the bacteriologists along a novel approach have been rewarded. Giles and Sangster[3] prepared agglutinating antisera from three strains of *Bact. coli* (biochemically identical with *Bact. coli No. 1*) isolated from three fatal cases and found that these strains were all serologically alike. Furthermore, 86 strains of *Bact. coli* out of 92 isolated from cases of primary gastro-enteritis were agglutinated by the specific antiserum, whereas this serological type of *Bact. coli* was isolated from only 2 out of 44 cases classified as enteritis secondary to some parenteral infection and from 3 out of 72 mild cases of infantile diarrhoea not treated in hospital. The organism was also found in the faeces of four infant contacts who 24 hours later developed diarrhoea; it disappeared from the faeces of affected infants as they recovered from the infection. These findings corroborate the earlier work of Bray,[3] and indeed the authors state that their organism closely resembles Bray's strain serologically, but in view of claims made from time to time for the aetiological relationship of other intestinal bacteria such as Morgan's bacillus, proteus and paracolon, *Staph. aureus*, and *Str. faecalis* a certain caution is necessary in their interpretation. The results obtained by other workers in this field will be eagerly awaited.

While the aetiology still remains unsettled, considerable advance has been made in the treatment of infantile enteritis. Acute vomiting and diarrhoea in an infant lead quickly to dehydration and an upset in the balance of body fluids which, if allowed to progress beyond a certain stage, become irreversible. The primary need therefore is to prevent or correct dehydration either by direct treatment and control of the diarrhoea and vomiting or by the replacement of lost body fluids. There have been many "cures" of gastro-enteritis aimed directly at the alimentary tract, either to reduce peristalsis, absorb toxins, or eliminate the infecting organism. In the last category the poorly absorbed sulphonamides—e.g., sulphaguanidine and "sulphasuxidine"—have proved disappointing, though they still have a vogue on the Continent. New antibiotics like aerosporin and streptomycin are now being used, and while in one study encouraging results have been reported with streptomycin[4] it is still too early to say what part these intestinal bactericides will play in the treatment of gastro-enteritis. Certainly they cannot be expected *per se* to correct severe dehydration and shock. Therefore effective treatment of the acute case of infantile enteritis must surely depend primarily on re-hydration in a way that will establish the proper balance of body fluids and osmotic pressure as quickly as possible and supply food until the alimentary system is again functioning normally. Subcutaneous or intraperitoneal glucose-saline may suffice in mild cases, but for the acutely dehydrated child, who is usually in a toxic or "shocked" condition, intravenous infusion of a Hartmann-glucose solution alternating with serum or plasma is essential.

The setting up of an intravenous drip and its maintenance for two or more days require experienced doctors and nurses. It has become the firm conviction of those engaged in the day-to-day treatment of infantile enteritis that satisfactory results will be obtained only in

[1] *J. Hyg., Camb.*, 1946, **44**, 480.
[2] Ibid., 1948, **46**, 1.
[3] *J. Path. Bact.*, 1945, **57**, 239.
[4] James, U., and Kramer, I. R. H. (with statistical analysis of results by P. Armitage), *Lancet*, 1948, **2**, 555.

special units with adequate accommodation—preferably as single-bedded rooms, for relapses from cross-infection are not uncommon—and enough trained nurses in the charge of an experienced medical officer. The organization of such a unit is described by Dr. M. B. Alexander elsewhere in this issue, and the results obtained—22 (8.5%) deaths among 258 dehydrated infants under one year of age—are a tribute to the teamwork of medical and nursing staff under conditions that were by no means ideal Dr. Alexander makes the interesting point that the case mortality in the four consecutive six-monthly periods following the establishment of the unit was respectively 11.3%, 2.4%, 2.4%, and 3.5%, which suggests that treatment becomes more efficient after the nurses gain experience in the handling of cases. When these results are compared with the fatality rates of 30%–60% which are still being experienced in different parts of the country and with the tragic reports of even higher rates in institutional outbreaks, the need for properly organized units for the treatment of gastro-enteritis becomes obvious. Large established units which could give experience to a maximum number of doctors and nurses in the minimum of time might be used as training centres for the medical officers and sisters who are to take charge of new units. They would thus become conversant with the organization and management of a going concern. Alternatively, a demonstration team could be sent to a hospital to help in establishing a new unit and to initiate medical and nursing staff into the intricacies of intravenous drips and the like. The newly organized flying squad at the Sick Children's Hospital, Great Ormond Street, has been written up in the lay press as something akin to a fire brigade, ready to fly at a moment's notice to quench some conflagration in the shape of an outbreak of gastro-enteritis. An equally useful function may be to act as a demonstration team by taking over the treatment of gastro-enteritis for some weeks in a hospital where it is proving troublesome, and thereby training the resident staff in the proper handling of this disease. We understand that the flying squad would regard this s one of its main purposes, and it is to be hoped that hospitals which are experiencing high fatality rates among infants admitted with diarrhoea and vomiting will make use of this service. The family doctor will play his part by not sending patients with mild dietetic disorders to hospital and by doing all in his power to encourage the continuance of breast-feeding when babies are reared in conditions where the standards of household hygiene and infant care are poor.

O RUSSIA! O MORES!

In a review in *Nature* in 1946[1] Professor Eric Ashby wrote: "As to the present state on the new genetics in the Soviet Union, it is safe to assume that Lysenko's school is well past its zenith." A year later, however, the Russian geneticist Zhebrak was severely taken to task by *Pravda* for daring to express his own views of Lysenkoism. "Zhebrak," *Pravda* pontificated, "as a Soviet scientist should have unmasked the class meaning of the struggle which is taking place around questions of genetics. But, blinded by bourgeois prejudices, by detestable fawning on bourgeois science, he has adopted the attitude of the enemy's camp." Reviewing the situation in a leading article in these columns in the *Journal* of Oct. 18, 1947, we observed that this attack by *Pravda* suggested "that genetics is once again becoming an acute political issue in the U.S.S.R." And now a year later the Lenin All-Union Academy of Agricultural Sciences has declared that the genetics of Lysenko is the only genetics that may be taught in Soviet institutions,[2] a decision which a special correspondent in *The Times*[3] says "has shocked scientists all over the world." Sir Henry Dale, O.M., President of the Royal Society from 1940 to 1945, has made his protest by resigning his honorary membership of the Academy of Sciences of the U.S.S.R. Sir Henry points out that it is clear from Lysenko's own statement "that his dogma has been established and enforced by the Central Committee of the Communist Party as conforming to the political philosophy of Marx and Lenin." Sir Henry Dale might have added as a footnote to his letter a statement made by Professor J. B. S. Haldane to a correspondent of the American magazine *Time*: "I don't think a political body should decide scientific theories."[4]

Dreary conformity to the deadening dogmas of the Marxist faith is being imposed on those unfortunate countries now being absorbed into the Russian Empire. This year the Czechoslovak medical association was decreed out of existence, and now doctors, nurses, and midwives in the Polish medical schools are obliged to take courses in dialectical materialism. The Polish Vice-Minister of Health has declared that "the philosophy of Marxism is an indispensable tool in the practical and scientific work of a physician." The basis of Polish medical schools, it is declared, must be "class consciousness and systematic political training."[5] As an example of how the Russian medical man has to pander to the Russian propaganda machine we may note the observations made by Professor S. A. Reinberg in an article[6] on mass radiography in the Soviet Union. Professor Reinberg is Director of the Molotov Central Institute for Scientific Investigation in Radiology. He notes that the number of radiography examinations made is "still less than those of some foreign series," and then goes on to explain this away by stating: "There is no doubt that these pretentious figures published in foreign countries constitute only a thinly disguised cheap advertisement." Mass radiography, says Professor Reinberg, "by its very nature is foreign to the ideas and training of the medical faculty in capitalist countries. The idea of radiography of large masses of workers does not correspond to the interests of a bourgeois society. . . ." This kind of nonsense, and the tragedy of death and exile of Soviet geneticists, must, we are sure, be silently condemned by countless men of medicine and science in the U.S.S.R. With them we must hope that the day is not far off when freedom to think and to speak and to write is once more restored to a great people.

[1] 1946, 158, 286.
[2] *Time*, Sept. 27, 1948.
[3] Nov. 6, 1948.
[4] Sept. 27, 1948, p. 34.
[5] *B.B.C. News Bulletin*, Nov. 3, 1948.
[6] *Problemi Tuberkuleza*, 1948, No. 5, 42–48.

TEMPERATURE RECORDING

In 1620 Sir Francis Bacon wrote, "Let further inquiry be made into the different degrees of heat in different parts and limbs of the same animal."[1] But very few studies of skin temperature were made till the nineteenth century, when Davy[2] investigated the problem in 1814. Others followed him, using the mercury thermometer, and later Becquerel

[1] *Novum Organum*, 1620. Book 2, Aphorism 13, No. 12.
[2] Quoted by Cobet, R., *Ergebn. Physiol.*, 1926, 25, 440.

and Breschet[3] in 1835 and Geigel[4] at Würzburg made more accurate studies with the thermocouple. Geigel took measurements in four different areas and compared them with the rectal temperature taken simultaneously. He showed that during a rigor the surface temperature falls while the rectal temperature rises, the difference between the two readings therefore increasing. In 1931 Fritz Talbot[5] published his monograph on skin temperatures in children. Using the thermocouple he made the following observations: variations occur in the temperature at any given spot on the body within five-minute periods; there are considerable differences in the temperature of the skin in different parts of the body, the temperature of the trunk being highest, that of the face next, and of the extremities lowest; the surrounding temperature has a considerable effect on the skin temperature, the cooler the room the cooler being the temperature of the skin; there is a rapid loss of heat from the surface of the body on exposure; exercise causes a preliminary fall followed by a rise of skin temperature; and other factors such as sleep and clothing affect the temperature. He confirmed the well-known fact that the rectal temperature is higher than that of the skin, and more recently[6] he has shown that the rectal temperature varies with the depth of insertion of the thermometer. It should be inserted 5 cm. in infants; the reading at 14 cm. will be 0.2–1.3° C. higher than if it is inserted 2–6 cm. It is worth noting that the rectal temperature may not always be higher than the oral. Rappaport[7] described a series of 25 patients whose rectal temperature was normal but in whom the oral temperature was raised persistently above normal. The reasons for this unusual finding are obscure.

Elsewhere in this issue Professor Alan Moncrieff and Dr. B. J. Hussey describe some simple experiments with the ordinary mercury thermometer. They mention the well-known fallacy of the "normal" axillary temperature: the child is chilled in the out-patient department, but when he has been warmed in bed a raised axillary temperature may be recorded within half an hour of his admission to the ward. The ordinary half-minute thermometer placed in the axilla gave a reading 0.4–1.4° F. (0.22–0.78° C.) higher after five minutes than after two minutes. Because of the unreliability of readings taken in the axilla or groin the authors advocate that these sites should no longer be used for temperature recording, and they recommend that temperatures should be taken rectally in all children under five years and by the mouth after this age. Allowance must of course be made for the fact that the rectal temperature is normally higher than that in the mouth. With proper technique the risks of cross-infection are small. Green and Penfold[8] recently investigated the risk of transmitting infection by hospital thermometers, and paid special attention to the efficiency or otherwise of the antiseptic solutions in common use. They obtained on culture up to 40,000,000 organisms per ml., including *Bact. coli*, from glycerin of thymol solution into which thermometers had been dipped on the nurses' rounds, but no organisms from 1 in 20 phenol. Every child should have its own thermometer kept in a safe antiseptic solution above the bed or in a cupboard; it should be sterilized when the child is discharged.

Taking the temperature is a simple procedure—so simple that frequently insufficient care is taken, particularly in the choice of site, in inserting the thermometer properly, in leaving it in for a sufficient length of time (at least two minutes for a half-minute thermometer), and in sterilizing it after use. We are prepared for technical errors in complicated laboratory tests, but often forget the possibility of errors in simple bedside procedures.

[3] *Arch. Mus.*, 1835, **1**, 385.
[4] Quoted in Albutt and Rolleston's *System of Medicine*, 1905, **1**, 834. London: Macmillan.
[5] *Amer. J. Dis. Ch'ld.*, 1931, **42**, 965.
[6] Brennemann's *System o, Pediatrics*, edited by I. McQuarrie, 1948, **1**, Ch.5, 3. New York: W. F. Prior.
[7] *Ann. intern. Med.*, 1946, **25**, 1.
[8] *Lancet*, 1947, **2**, 89.

THE MILK BILL

Last week the Government introduced into the House of Lords the Milk (Special Designations) Bill. This will give the Minister of Food power to specify areas in which only specially designated milk may be sold retail or supplied under the milk-in-schools scheme. The approved designations in England and Wales will be T.T. (certified) milk, T.T. milk, accredited milk (derived from a single herd), pasteurized milk, and sterilized milk. In Scotland the designation "standard" takes the place of "accredited." Descriptions of these milks and the conditions which must be complied with before they can be so designated will be set out in Milk (Special Designations) Regulations.

In a recent annotation[1] we drew attention to Lord Bledisloe's observation that Great Britain, with about 40% of her cattle infected with tuberculosis, has the worst record in this respect of all the countries in Western Europe. Milk must be made safe for the public to drink, and at the same time the health of dairy herds must be improved. The Minister of Agriculture bears responsibility for the latter task, but the Minister of Food under the new Bill is now to be given power to ensure that only heat-treated milk or milk from disease-free animals may be sold in certain areas of the country. It has often been suggested that the public might be misled by the term "accredited" into thinking that milk from accredited herds must be "safe," and it is encouraging to see in the new Bill that recognition of accredited and standard milk as designated milks will be restricted to a period of five years from the commencement of the Act, and that in the meantime the designation will apply only when the milk concerned comes from a single herd. Many would wish that it might have been possible to dispense with this designation immediately. In order that the Minister of Food may be in a position to carry out the requirements of the Bill he is to have power to install and operate heat-treatment plants wherever he may decide they are necessary, or he can arrange for the work to be done by local authorities.

Under Defence Regulation 55G (now to be revoked) the Government has had powers for the past five years to impose limitations similar to those proposed in this Bill. So far as we are aware, no area of the country has yet been specified as an area in which only designated milk may be sold. Shortage of heat-treatment plant and of skilled labour cannot be the only reasons for this deplorable gap, during which many lives have doubtless been lost and much illness caused by infection with bovine tubercle bacilli. With the powers proposed in this Bill the Minister of Food should at last be in a position to correct what is recognized by the medical profession to be a serious hygienic defect in this country.

The Goulstonian Lectures on "The Cardiology of Old Age" will be delivered by Dr. C. J. Gavey, F.R.C.P., before the Royal College of Physicians of London (Pall Mall East, S.W.) on Tuesday and Thursday, Jan. 11 and 13, 1949, at 5 p.m.

[1] *British Medical Journal*, 1948, **2**, 868.

LOYAL ADDRESS OF THE BRITISH MEDICAL ASSOCIATION

We print below the loyal address presented to His Majesty the King, Patron of the British Medical Association, on the occasion of the birth of a son to Her Royal Highness Princess Elizabeth.

To the King's Most Excellent Majesty :

The Humble Address of the President and Members of the British Medical Association

May It Please Your Majesty,

We, Your Majesty's dutiful and loyal subjects, the members of the British Medical Association distributed throughout Your Majesty's Commonwealth and Empire, humbly offer to Your Majesty, and to Her Most Gracious Majesty the Queen, congratulations on the birth of a son to Her Royal Highness the Princess Elizabeth, Duchess of Edinburgh.

Our gladness on this auspicious occasion is joined with a fervent hope that the young Prince may enjoy length of days, richly blessed with happiness and health.

With our heartfelt felicitations we beg to express to Your Majesty our high and affectionate regard and to assure Your Majesty of our unceasing fidelity and loyalty to Your Majesty's Throne and Person.

Signed on behalf of the British Medical Association:

LIONEL WHITBY, *President.*
H. GUY DAIN, *Chairman of Council.*
E. A. GREGG, *Chairman of the Representative Body.*
A. M. A. MOORE, *Treasurer.*

Reports of Societies

PREVENTION OF ACCIDENTS

The prevention of accidents was the theme at a meeting of the Section of Epidemiology and State Medicine at the Royal Society of Medicine, held on Nov. 1, with Sir Allen Daley in the chair. Mr. W. GISSANE, surgical director of the Birmingham Accident Hospital, discussed the incidence, severity, and after-effects of accidents, domestic and industrial, with special reference to the provision of efficient treatment, and showed that despite some improvement there was no room for complacency. He thought that patients with accidental injuries endangering life were too often given treatment too late or by medical personnel inadequately trained for the task. Suitable structural arrangements and modern equipment were required in hospitals for this purpose, and team-work was necessary for even reasonably good results. Adequate after-treatment should be given by the same team. As a result, crippling deformities which in the past lowered industrial efficiency were rare, and the effect of treatment was greatly improved when patients received rehabilitation specially designed to fit them for a particular task or occupation. He felt that, while prevention of accidents might be a very difficult problem, the prevention of the crippling results was something which could be tackled immediately.

Dr. LEONARD COLEBROOK gave an interesting analysis of 736 cases of accidents treated in Birmingham. Of these, 504 occurred in the home (all ages) and 232 in industry. The fatality of the home accidents was six times that of the industrial accidents, and the average stay in hospital was approximately 25% longer—due largely to the more serious accidents occurring in the home, especially burns and scalds in children and old people. One-third of the patients burnt at home were epileptics, and the burns were caused mainly by tea and other hot liquids, coal fires, electric and gas fires, baths left unattended, and inflammable liquids. Electrical faults and conflagrations constituted a very small part. The need for prompt admission to hospital without prior treatment apart from protection of the damaged tissues was stressed as part of the aseptic technique.

The Danger of Carelessness

Dr. C. A. BOUCHER stated that more than 8,000 persons were killed every year in England and Wales as a result of accidents at home and in everyday pursuits. Six thousand of these were in the home, which represented about two-fifths of the total of all fatal accidents, including road accidents, transport accidents, mine accidents, etc. There had been no fall in the figure since 1938. On the contrary, in the age groups under 5 and over 65 there had been an increase. In 1945 one-fifth of all fatal accidents occurred in children under 5 and about half in persons over 65, falls being the main cause in the latter group. Structural defects in the home or faulty house design were not considered to be a major cause. On the other hand, overcrowding in the home was a definite factor, especially in the case of accidents affecting children. Dr. Boucher laid emphasis on carelessness and neglect about the dangers existing in the home and the use of faulty appliances which looked tempting and cheap in the shops and could be purchased so easily by the non-discriminating person. He felt that there was a great lack of awareness among the people concerning the extent of this problem. There was also indifference, when it was realized that the majority of these accidents were preventable. He believed that there were at last signs of activity directed towards the prevention of home accidents. The Royal Society for the Prevention of Accidents had shown great energy and imagination in drawing the attention of the public to dangers in the home. This society was the chief medium through which educational work was initiated. There was also an Inter-departmental Committee on Accidents in the Home set up by the Home Secretary in 1947 to co-ordinate the work of departments in preventing accidents in the home, and a Domestic Accidents Panel of the Building Requirements Subcommittee of the Ministry of Works was already at work.

In Dr. Boucher's opinion the most hopeful course was to use education and propaganda rather than legislation. He quoted the ineffectualness of the provision in the Children and Young Persons Act, 1933, in which it was decreed that any person over the age of 16 having the care of a child under the age of 7 who was killed or seriously injured because of an insufficiently guarded open firegrate shall be liable to a fine of not more than £10. Nobody could penalize a woman, particularly a mother, for so tragic an accident. He thought that the aim of education should be to make the public critical of goods which they bought in the hope that in time public opinion would demand safe articles; in the schools teachers could help by giving suitable instruction to children.

Need for Reliable Statistics

Dr. PERCY STOCKS, mentioning the work being done in America to reduce home accidents, thought that general appeals were unlikely to succeed and that each cause of accident would have to be attacked in detail. A necessary prelude to such action was to obtain good statistics, and the General Register Office was doing three things to that end. Coroners' certificates were providing more detail than hitherto of the circumstances of fatal accidents and the places where they took place. After lengthy discussion with experts in the U.S.A. a classification both by nature of injury and cause of accident had been grafted into the International List, which was now designed for morbidity as well as mortality statistics. This contained definitions and enough detail of grouping to satisfy anyone. The teaching hospitals had in the past paid little attention to causes of accidents, but now they were being asked to do so in the interests of prevention. It was hoped to secure sufficient information about in-patients during 1949 to provide some useful statistics. He believed that the hospitals could in that way make an important contribution to the eventual reduction of many kinds of accidents.

Dr. BRIAN STANFORD gave a preview of a new film-strip lecture addressed to persons who have the care of children. This showed the main causes of home accidents and the ways in which they could be prevented. Mr. JOHN BUNYAN described a bag which could be used to cover a limb and thus provide adequate protection until the injured patient reached medical care.

Correspondence

Treatment of Placenta Praevia

SIR,—The excellent paper by Mr. W. G. Mills (Nov. 20, p. 896) in which 100 cases of placenta praevia are recorded with no maternal deaths and a foetal and neonatal mortality of 16.5% provides further convincing evidence of the value of expectant treatment of this disease, and shows that it can be carried out, in proper surroundings, with safety to the mother and increased chances of survival for the child. I have advocated this treatment for 20 years or more, and described it in the first edition of my book, *Antenatal and Postnatal Care* (1935), London, and during that time it has been practised in the obstetric unit of University College Hospital, London.

It is encouraging to note that Mr. Mills adopts the newer classification of placenta praevia into four degrees in preference to the older and confusing one into lateral, marginal, etc. On one point, the treatment, I have advocated views differing from his—i.e., that once a "warning" haemorrhage has occurred after the 28th week the patient should be kept in hospital under observation until delivery. The majority go into labour spontaneously at term, deliver themselves without further bleeding, and go home without any exploration of the lower uterine segment having been made, and consequently without the nature of the haemorrhage—whether unavoidable or accidental—having been accurately ascertained. Only a speculum has been passed shortly after admission to view the cervix and exclude any cervical lesion. In this annual report of University College Hospital such cases are classed as "haemorrhage of unknown origin," and four varieties of antepartum haemorrhage are included: (1) haemorrhage certainly due to placenta praevia, in which the placenta has been *felt* or *seen* in the lower segment; (2) those in which it is not felt or seen there (accidental haemorrhage); (3) those due to haemorrhage from extra-placental sites, usually the cervix; and (4) those in which it is not known if the haemorrhage is accidental or unavoidable. The last is by far the largest class in each annual report. In other words the report is an honest one not attempting to classify where accurate classification is impossible.

Some other hospitals have already adopted this classification, but it is disappointing to find that in the recently published "standard" form for annual reports (*J. Obstet. Gynaec. Brit. Emp.*, 1948, 55, 478) drawn up by a committee of the Royal College of Obstetricians and Gynaecologists the old one into three varieties only is adopted. Frankly I am always suspicious, to put it mildly, of any report that classifies all cases of antepartum haemorrhage as due either to placenta praevia or to premature separation of a normally situated placenta (accidental haemorrhage). Into what class the case is put so often depends on the whim of the house-surgeon or registrar, or on examination of the placenta after its delivery—an entirely unreliable guide, as experience proves.

The expectant treatment often means that patients have to be kept in hospital for several weeks before delivery, but all who have practised the method will agree that it is well worth while. It makes, too, a great demand on antenatal beds, but bed scarcity is no reason for condoning inadequate antenatal care. If we do not, in the interests of our patients, demand more antenatal beds we shall certainly never get them.—I am, etc.,

London, W.1. F. J. BROWNE.

SIR,—Whilst congratulating Mr. W. G. Mills on his results of treatment of placenta praevia (Nov. 20, p. 896), I should like to point out that his figures are not comparable with other statistics. Vaginal examination under anaesthesia is now practised in some obstetric centres in all cases of antepartum haemorrhage after the thirty-sixth week of pregnancy unless the bleeding is of extraplacental origin. I gather that this is the routine at the Birmingham Maternity Hospital.

Such a practice results in accuracy of diagnosis and successful results, but it also gives a diagnosis of placenta praevia to a proportion of that large group of cases which Professor F. J. Browne includes under the heading "antepartum haemorrhage of unknown origin." Now this group comprises those cases of haemorrhage which are mild, and the majority of them end in a spontaneous and successful delivery. If cases of placenta praevia from this group are added to statistical summaries it will "improve" the maternal and foetal death rates. This may in part account for the apparent success of Mr. W. G. Mills's results.—I am, etc.,

London, W.C.1. J. G. DUMOULIN.

Primary Post-partum Haemorrhage

SIR,—I have only just seen the *Journal* of Oct. 23, but I would like to comment on the article by Drs. James B. Joyce and G. Gordon Lennon at p. 740 of that issue. It is stated, "We believe it is better to go to the highest point of the placenta and start separation from above downwards." It seems to me that a placenta situated on the posterior wall would be extremely difficult to remove by this method. It would mean the operator working with an acutely flexed wrist in the confined space of the uterine cavity. This would create a tremendous mechanical disadvantage, as well as causing the hand to become quickly exhausted if the operation takes longer than anticipated. It would also mean introducing far more of the hand, wrist, and forearm into the uterine cavity than is necessary.

The reason given in support of this method is that the uterus can retract immediately on the open sinuses. Surely with adequate control of the fundus with the external hand there is not much chance of venous bleeding occurring while the operation is actually being carried out. With an operation such as this, in which one has always to work alone and in the dark, it seems a pity to confuse the first-time operator (who is often considerably in awe of the whole procedure) with theoretical directives, however explicit. All this apart from the fact that it is much easier to remove a placenta from below upwards.

The authors include interesting quotations from obstetric forebears; consequently it seems doubly hard on Credé that his name should be omitted deliberately, and so uncere-moniously, from Part II. The reason given, that he never awaited the natural separation of the placenta before applying his method, seems irrelevant. Credé may have been an impatient man, but surely the technical method is the same at whatever moment after delivery of the child it is carried out —I am, etc.,

Toronto, Canada. ROBERT A. H. KINCH.

Breast-feeding

SIR,—I read with interest Dr. Enid L. Hughes's article on breast-feeding (Sept. 25, p. 597). In the fishing community of Newfoundland marriage, having babies, and breast-feeding are looked upon as natural for a woman. As a result the mothers have a very much easier time while having their babies than do their sisters in England. Dr. Grantly Dick Read's teaching is not needed here, for mothers accept their labour as something essentially normal, not something to be feared and struggled against as in England.

Likewise with breast-feeding. In the houses there is rarely any possibility of privacy, but the mother feeds her baby in full view of her family and any visitors who happen to be present. While waiting to see the doctor it is quite common for the mother to give her infant a "meal." At least 95% of the babies are breast-fed, and a woman who does not breast-feed is looked down on.

Unfortunately the above does not obtain in the towns, where the situation is the same as in England—giving birth is a "disease," and breast-feeding is carried on by a much smaller percentage. It would be of interest to conduct a survey of breast-feeding considering the social status of the family.—I am, etc.,

Burgeo, Newfoundland. M. G. JACOBY.

SIR,—The figures given by Dr. Enid L. Hughes (Sept. 25, p. 597) regarding failure of lactation raise the question how important this is. After 19 years in M. and C.W. work my views have changed, and I do not now regard artificial feeding as a disaster. On the contrary, I think the gains to mother and child are many. The mother is relieved of a function

which is an irksome tie and often detrimental to her health, and the baby, brought up on dried milk preparations fortified with vitamin D and minerals, plus cod-liver oil and orange juice, receives a diet lacking nothing needful for its optimum development. In fact, at the five-year-old routine school inspections I have often remarked that the children with the best teeth have been artificially fed and, contrariwise, that the worst dentition tends to be correlated with breast-feeding, particularly if this has been prolonged.

Moreover, the teaching that breast milk is a perfect diet is, we now know, fallacious and often results in the withholding from the infant of the necessary vitamins, iron, etc. We do not know the ideal length of time for breast-feeding, but it is probably much shorter than the fashionable 6 to 9 months. As for the loosening of the emotional tie between the lactating mother and her infant, this I think is ideological. I have never noticed any diminution of affection in the mother or of satisfaction in the infant when bottle-feeding is instituted. On the contrary, when the babe is partly breast-fed and partly bottle-fed the mother almost invariably says it prefers the bottle to the breast.

In short, during the puerperium and for a few weeks following I think breast-feeding is desirable if it can be achieved. But it is a primitive function which the majority of women find they cannot fulfil in the rush and bustle of urban life for any length of time.—I am, etc.,

Exeter, Devon. GRACE H. WALKER.

Ménière's Syndrome

SIR,—We have been interested in the work done by Mr. E. R. Garnett Passe and Dr. J. S. Seymour (Nov. 6, p. 812) in treating Ménière's syndrome by interrupting the sympathetic fibres from the stellate ganglion running along the vertebral artery, and in some cases by dividing the artery as well. They raised the problem of ligating the second vertebral artery if the condition should also develop on that side, and they suggest that the safety of this procedure should first be proved by animal experiment.

We have been doing some experimental work on blood flow in the circle of Willis using rabbits; of the readily available animals they have a circle most like that of man (cats and dogs are especially unsuitable for anatomical reasons). Ligation of both vertebral arteries has been done by other workers besides ourselves and does not result in death. Using a rapid coagulation technique modified from that of Franklin and Amoroso[1] we have confirmed earlier work that under normal circumstances the vertebral arteries supply the hind-brain and posterior-cerebral-artery territory. And we have shown that in this animal carotid and vertebral blood-flows meet and oppose each other without appreciable mixing in the posterior communicating arteries.[2] Any alteration in balance of pressure between the carotid and vertebral circulations causes the posterior communicating artery to function as an anastomosis, and blood flows along it.

In man the condition of the posterior communicating arteries, therefore, would appear to be all-important in the main problem under consideration. Normally these are relatively smaller than in the rabbit, but they show great variation. Dandy[3] found that they were "normal" in less than 50% of 1,033 cases; and in 3% of 1,603 cases they were both absent. Fetterman and Moran[4] found absent or threadlike posterior communicating arteries in 23% of 200 cases, averaging 59 years of age. These cases showed a higher incidence (31.8%) of cerebral softening than cases without these defects (23%). Because of these variations it would be unwise, regardless of the results of animal experiment, to attempt to predict the results in man of bilateral vertebral ligation, especially in older patients. Under rarer circumstances the structure of the circle of Willis might render even unilateral vertebral ligation dangerous. To investigate the presence and patency of the posterior cerebral artery in any individual arteriographic studies might help, or else close observation could be made at operation during bilateral vertebral compression.

A further unknown factor is the possibility of thrombosis. The injection of small doses of thrombin into one vertebral artery has invariably resulted in death of the animal from what appears to be respiratory followed by cardiac failure, whereas larger quantities injected into the internal carotid artery did not cause death. While the thrombosis resulting from intravascular injection of thrombin is hardly comparable to that which might follow the ligation of a vessel, we think this observation worthy of record. Although much of this is theoretical as regards man, we feel that the factors mentioned should be appreciated if interference with the vertebral artery, whether by ligation or by injection of radio-opaque substances, is to become more common than hitherto.—We are, etc.,

DONALD A. McDONALD.
London, E.C.1. JOHN McE. POTTER.

REFERENCES

1 Oral communication, Physiological Society, March 23, 1948.
2 McDonald, D. A., Potter, J. M., *Proc. physiol. Soc.*, 1948. (In the press.)
3 *Intracranial Arterial Aneurysms*, 1945, p. 147, New York.
4 *Arch. Path.*, 1941, **32**, 251.

A Gastric Bezoar

SIR,—On Oct. 16 a man aged about 40 was admitted to the Waveney Hospital, Ballymena; he had had vomiting and stomach discomfort for about three weeks. He was of low-grade intelligence, and no clear and reliable history of his condition could be got from him. So far as we could find out he complained of a dull, aching pain in his epigastrium which radiated upwards, occasionally woke him up at night, and was relieved by a drink of milk. There appeared to be no previous history of stomach trouble. His appetite was good, and he had no loss of weight.

On examination by the H.S. when admitted his abdomen was noted as lax and mobile, with no tenderness or rigidity, and no abnormality was discovered. When examined next day by me I noted some abdominal distension and a certain amount of rigidity, making it impossible to palpate the abdominal organs clearly. On Oct. 17 he had a fractional test meal done, but there was great difficulty in getting an adequate specimen from the stomach at any time. Such specimens as were got showed no free HCl and diminished total acidity. On Oct. 18 he was *x*-rayed. On examination six hours after a barium meal the meal seemed to be in the normal position in the caecal region, but there was a faint shadowy picture of an enlarged stomach visible. On getting his second barium meal the stomach was shown considerably enlarged, with some thinning of the barium shadow in the pyloric and cardiac regions which could hardly be described as definite filling defects.

When I examined him on Oct. 21 before a meal he was quite relaxed, and I was able to feel a definite mass occupying the whole stomach area and freely movable. At operation on Oct. 22 I removed from inside his stomach a rolled mass of hay and straw weighing 1 lb. 12 oz. (793 g.). This was tightly rolled and matted in the pyloric region and looser and less compact in the cardiac region. His recovery has been uneventful save for a stitch abscess, and his only complaint at present is that he does not get enough to eat.—I am, etc.,

Ballymena, N. Ireland. J. ARMSTRONG.

Intussusception Due to Carcinoma of Colon

SIR,—Surely Mr. R. A. C. Owen (Oct. 30, p. 786) is wrong with regard to the rarity of intussusception in adults, interesting though his cases were, and that the opposite holds true, namely that they are too common to require reporting? If not, my first case, an ileo-ileal intussusception headed by a submucous lipoma, is hardly relevant to his article, but my second has its own interest.

The patient was a woman aged 49, and she was seen on July 16, 1940, with a history of increasing constipation. A mass was felt attached to the left corner of the uterus. She was later moved to the Staffordshire General Infirmary, and on July 26 a growth (histologically adenocarcinoma) of the sigmoid colon together with attached portions of the uterus and bladder wall was removed, using a modified Paul operation. She returned home healed and well in September, 1940, and nothing further was heard of the patient till March 20, 1945. At this time she had a sudden violent attack of abdominal pain and vomiting. This gradually settled down, and a subsequent barium enema gave a "normal" (i.e., indeterminate) result.

It was more than a year later that she was confined to bed with griping pains and constipation (May 7, 1946). She was treated expectantly with oil and magnesia, with fair apparent success. On

May 16 there was a relapse, and a vague tumour in the left upper abdomen appeared together with visible peristalsis. On May 17 blood was passed per rectum, and laparotomy showed the head of an intussusception from the mid-transverse colon to have just reached the splenic flexure. Reduction was easy, and a malignant growth was manifest. Search of the abdomen showed no distant secondaries nor any evidence of trouble in the sigmoid colon or pelvis, and the operation was completed by the formation of a caecostomy. On June 7 nine inches of the mid-transverse colon was excised centring on what proved again to be an adenocarcinoma, and continuity of the bowel restored by side-to-side anastomosis. The caecostomy shortly closed, and the patient returned home on June 25 and has remained well since.

A personal view is that the second carcinoma had a quite distinct origin from the first. The occurrence of pain, etc., fifteen months before its first discovery indicates a possible polypoid origin which might be common to the two, but no other polypi were manifest at the 1946 operation. However, the concurrence of, to my mind, two not very rare conditions, discrete colonic growths with intussusception of one of them, is unusual, and comment on either subject would be welcomed. —I am, etc.,

Stafford. G. I. WILSON.

Aortic Dissecting Aneurysms

SIR,—I read with interest the article on aortic dissecting aneurysms by Mr. D. P. van Meurs in your issue (Sept. 25, p. 599) which reached here this week, just two days after a discussion on the same subject had taken place at the weekly clinico-pathological conference at the Royal Hobart Hospital attended by resident medical staff, consultants, and local general practitioners.

The case under discussion was a man aged 65 whom I was called to see at 6 a.m. on Nov. 1, 1948. He had been seized with an acute abdominal pain on rising and had collapsed to the floor. On arrival I found him on the floor in a state of severe shock. He was extremely pale, sweating, but not dyspnoeic. He retched twice in my presence, but did not vomit. His radial pulse was of poor volume, rate 96, rhythm regular. The heart was not enlarged clinically, and the sounds were normal. Blood pressure was 80/40. There was rigidity of the abdominal musculature in all areas. I considered he was far too shocked for a case of perforated abdominal viscus and sent him to hospital with a diagnosis of dissecting aneurysm of the abdominal aorta. He revived somewhat after admission to hospital and was able to give more history. He had no relevant past history. The abdominal pain commenced soon after he retired, and it woke him at intervals during the night. He was seen after admission by a surgeon, who advised against laparotomy. The patient was then feeling much better and had only a dull ache in the abdomen. The abdomen was rigid all down the right side and out into the right loin. On the day following admission he still had a fair amount of pain. At 2 p.m. he began to sweat, went pale, and died in a few minutes.

Post-mortem examination showed a rounded aneurysm of the abdominal aorta about 3 inches in diameter situated just proximal to the bifurcation. The aneurysm had ruptured over a wide area anteriorly, and several pints of blood were present in the adjacent tissue spaces and in the mesentery and peritoneal coat of the intestines. The aneurysmal sac was adherent to the lumbar vertebrae, but there was no erosion. The inside lining of the aneurysm showed some calcification, much necrosis and stripping of the walls, and some slight tendency to longitudinal wrinkling. The proximal aorta and the iliac arteries were arteriosclerotic, with calcareous plaques. Apparently the aneurysm had been leaking all night, and the effort of rising had caused further dissection with terminal rupture 36 hours later.

I am indebted to Dr. J. C. Laver, superintendent, Royal Hobart Hospital, for the clinical notes after admission, and to Dr. Campbell Duncan, Commonwealth Government pathologist, Hobart, for the post-mortem report.

—I am, etc.,

Hobart, Tasmania. A. W. O. YOUNG.

Bronchial Asthma and Thiopentone

SIR,—Dr. Brian D. L. Johnson's letter (Nov. 6, p. 837) calling attention to the fact that bronchial spasm can follow the administration of pentothal to a patient suffering from bronchitis and asthma would have been more convincing had he described his induction in greater detail. That type of case can be safely anaesthetized with thiopentone provided due care and attention are given to the underlying pathology. Most or all of the lung tissue is affected by bronchitis-emphysema and asthma in varying degree; probably there is bronchiectasis and residual sputum in addition. Secondary deposits are also possible in the type of case described by Dr. Johnson. Such a patient is quite unfit for the strain of any athletic performance and she is equally unfit for a rapid induction with drugs that will also cause respiratory depression. Such cases are sick patients and must be anaesthetized with considerable respect. For some years I have adopted the following procedure in all bad risk cases and without exception have always had most satisfactory results.

The patient's arm is put on a splint. A syringe of 20 ml. of thiopentone 5% is taken and the needle inserted into a vein. The syringe is strapped on but no injection is made. With some 8 litres per minute of oxygen running through a Boyle's machine a mask is placed near the patient's face. He is told what is happening—that the oxygen is to help his lungs, etc., that he will not go to sleep yet and that he will be told when he is to be put to sleep. I have never seen any anxiety follow this procedure. While talking to the patient the mask is lowered on to his face; frequently the colour improves but in any case at the end of about two minutes or less 1 ml. only of thiopentone is injected and the patient is told that he will go to sleep in the next few minutes. Rarely does this dose upset respiration, but if it does no further injection must be made until good movement has been restored. Some 30–45 seconds later a further 1 ml. is injected and the result observed. The injection can now be continued at an increased speed, but slowing of respiration must not be ignored. After the first 5 minutes, and usually after 0.5 mg. thiopentone has been given, the oxygen can be reduced and N_2O added. The mixture should be 50% at first, and the O_2 content should not drop to say 20% in under 10 minutes, though the operation can have started before then.

Bronchial spasm, it it occurs, will be preceded by a bout of coughing. Further steps that can be taken to avoid this coughing are: (1) Do not risk inserting an artificial airway at too light a plane of anaesthesia. Be sure that an adequate plane has been reached before this is done. (2) If further anaesthetic agents are to be given, remember that ether will set up violent coughing if not used carefully. Chloroform is at its best in this type of case and makes a good stepping-stone to ether.

Finally, though one has had a smooth induction and anaesthetic a stormy recovery may follow. Long bouts of coughing and straining can be separated by periods of breath holding. This tends to produce a blue patient, perhaps in spite of the administration of oxygen. My conclusions from this, if the patient has a really chronic chest, are as follows: (1) Use local if reasonable—e.g., for suprapubic drainage. (2) Limit the thiopentone to 1 g., and a quicker recovery will ensue. (3) Early removal of artificial airways is important. (4) Do not leave the patient lying flat if coughing is occurring. They are better well propped up.

The administration of oxygen before inducing anaesthesia is too little practised. I have twice previously described my technique in the *Journal* and know how smoothly bad risk cases can be induced in this way. It is also very regrettable but equally true to say that many patients are inadequately oxygenated when they go blue from any cause. This crime—for to me it is none other than culpable negligence—is by no means confined to the junior anaesthetist and may even be committed in teaching hospitals.—I am, etc.,

Southsea. H. B. C. SANDIFORD.

Pulmonary Rheumatoid Disease

SIR,—The paper by Drs. Philip Eilman and R. E. B. (Nov. 6, p. 816) raises several interesting points. The first is that, whatever causes pulmonary fibrosis in rheumatoid disease, it is a rare finding in such cases. Skiagrams of the chest are usually normal in this disease. In view of this we feel justified in reporting a case we have at present under our care.

A clerk, aged 43, first developed symptoms of rheumatoid arthritis in 1934, in his feet at first and later in his knees. Under physiotherapy the disease remitted, only to reappear in more active form in 1941 in his hands, wrists, and elbows. A further relapse occurred in 1947, when his shoulders became affected. In 1943 he became aware of the onset of swelling and redness of the finger-tips, which subsequently remained swollen and pink. In 1945 he had a short sudden attack of dyspnoea with palpitations. Skiagrams and electrocardiographs were normal. One year later a sharp pain of pleu

type appeared at the right base, abating within a fortnight. Treatment had been gold, vaccines, lactic acid intra-articular injections, and physiotherapy.

His previous history was as follows. In 1928 he was treated for thyrotoxicosis by iodine and deep *x*-ray therapy. The last treatment was given in 1932. The condition subsided and has never relapsed since. Total dental extraction was performed in 1934. He gave no past history of chest trouble and had never been exposed to dust hazards. His family history was irrelevant. An elder brother also had thyrotoxicosis.

On examination he was found to have bilateral exophthalmos; there were no other signs of thyrotoxicosis. His fingers were markedly clubbed, the terminal phalanges being bright pink in colour. The cardiovascular system was normal: B.P. 120/80. The chest was barrel-shaped: maximal expansion 2¼ in. (5.6 cm.). A few fine basal rales were present. Signs of advanced rheumatoid arthritis were present in elbows, wrists, hands, knees, and the left shoulder. The Wassermann reaction and gonococcal fixation tests negative. Blood count normal. Haemoglobin 95%. Electrocardiograph normal. Serum cholesterol 214 mg. per 100 ml.; plasma proteins 6.4 g. per 100 ml. (albumin 4.2 g., globulin 2.2 g.). E.S.R. (Wintrobe) varied between 13 and 34 mm. Non-protein nitrogen 22 mg. per 100 ml., plasma uric acid 3.7 mg. per 100 ml. Serum alkaline phosphatase 9 units. Thymol turbidity 2 units; thymol flocculation 1+; serum colloidal gold negative. Twenty-four-hour specimens of urine contained 1.2 g. creatinine and 0.2 g. creatine.

Biopsy of the skin and deltoid muscle was reported on by Dr. A. W. Morgan as follows: "The capillaries of the cutis vera are rendered prominent by congestion and endothelial swelling; also by a mild lymphocytic infiltration of the perivascular lymph spaces. The portion of muscle shows little of note in the fibres themselves and no fibrosis. There is, however, a focal infiltration of round cells between the fibres and a slight degree of sarcolemmar nuclear proliferation."

Skiagrams showed typical rheumatoid changes in knees, hands, and wrists. The cardiac outline was normal. The lungs showed a fine reticulation in lower and middle zones, with coarser nodulation in the outer quadrants of the middle zones. Progress was slow but uneventful. The patient is still under observation as an out-patient after spending four months in the ward.

The case is still *sub judice*, but we consider it worth reporting in the light of Ellman and Ball's report. The history and clinical and radiological findings do not suggest pulmonary tuberculosis or bronchiectasis. Polyarteritis and sclerodermia were considered as diagnoses but were rejected. We have for some time considered him to be "rheumatoid disease of the lung," as the pulmonary changes appeared during the course of the disease, were not accompanied by symptoms, and did not appear due to other causes. The patient, an intelligent man, is certain that finger clubbing appeared first in 1943 between two acute relapses in the rheumatoid condition. In view of Ellman and Ball's report we feel inclined to adhere to our diagnosis and advance this, tentatively, as a further report of "pulmonary rheumatoid disease."—I am, etc.,

London, S.W.1. F. DUDLEY HART.

Hereditary Haemorrhagic Telangiectasia

SIR,—I was most interested in the article written by Dr. C. P. Petch (Oct. 30, p. 785) on this condition, sometimes known as Osler-Render-Weber's disease (Parkes Weber first stressed its hereditary nature by describing a family in 1907, and he reviewed the literature in 1924). In reviewing some of the literature (with a report of a rather unusual case with a four-generations pedigree) in the *Journal* of March 31, 1945 (p. 440), I got the impression that the disease was not quite as rare as the number of actually recorded cases might indicate. Clinicians like Hurst and Plummer stated this, and Price goes further to say that the condition may be as common as haemophilia. Certainly the difficulty has been to distinguish clearly this condition from disorders like purpura, haemophilia, pseudo-haemophilia, and so on.

The criteria could perhaps be enumerated as follows: (1) It is hereditary, showing as a Mendelian dominant and not sex-bound; (2) the presence of telangiectases; (3) the tendency to haemorrhage; and (4) the multiplicity of lesions. The disease usually runs a fairly benign course like most Mendelian dominants, otherwise possibly the gene would sterilize itself. Its fairly good prognosis was borne out by Campbell,[1] who did not agree with Osler's view.[2] The telangiectases may be present anywhere on the skin, the mucosa of the alimentary canal and the bronchial tree, or even in the capillaries of the brain, but usually they are found in the nose. The bleeding is sporadic, epistaxis being the common feature. Hess's test is negative: it is this fact which would throw doubt on the cases reported by Singer and Wolfson[3] in 1944.

Finally I would like to mention that I was not quite certain at what stage the patient reported by Dr. Petch had attempts at cautery done. Hurst and Plummer[4] described a method of immediate control by the inflated-fingerstall device; when the bleeding settled cauterization of the larger naevi was performed. This has been found a most efficient method of treatment, though admittedly a new crop of naevi are liable to occur.—I am, etc.,

London, S.E.1. D. CAPPON.

REFERENCES
1 *Lancet*, 1944, **2**, 502.
2 *Principles and Practice of Medicine*, 1936, New York.
3 *New Engl. J. Med.*, 1944, **230**, 637.
4 *Guy's Hosp. Rep.*, 1932, **82**, 81, 87, and 96.

H 11 in Malignant Disease

SIR,—Case 5 reported by Mr. J. H. Thompson and Dr. G. J. W. Ollerenshaw (*Journal*, Nov. 6, p. 835) and the third case referred to by Dr. H. Josephs (Nov. 13, p. 876) appear to be identical with a case recently under my care. In both the above references the assumption is made that here was a case of carcinoma of the rectum with secondaries in the liver which disappeared as a result of H 11 therapy. Every doctor who accepts this assumption will feel that a very good case has been made out for this therapy in cancer. The history of the case known to me, however, is that the nodules in the liver were not typical secondary carcinomatous nodules. They were described by the original operator as "very small nodules," and in a personal communication he tells me that "he would not now regard such nodules as cancerous." Every surgeon of experience must have met similar small nodules which on section turned out to be fibromas or hepatomas or small cysts or other benign lesions. The patient has done very well, and I do not deny that H 11 may have been beneficial, but it has not been proved that such a striking and unusual effect as the disappearance of liver secondaries has occurred.—I am, etc.,

Birmingham. FAUSET WELSH.

Pensions for Diabetics

SIR,—I am rather surprised at the somewhat unsympathetic attitude adopted by Dr. R. D. Lawrence (Nov. 13, p. 875) in view of his well-known interest in the welfare of the diabetic and of his work for so many years for those who have this handicap. As he points out, recruits found to have glycosuria were most carefully excluded from service with the Forces unless this condition was proved to be of the harmless variety. It was therefore to be expected that the incidence of diabetes among serving members of the Forces would be low. But nevertheless many were admitted with a diabetic heredity; in these the exigencies of war might easily precipitate a diabetic state which might never otherwise have occurred, or antedate its onset.

A case of this type came under my own notice. This man, whose mother died of diabetes, was passed fit for service, trained for the commandos, put in strenuous service in Syria, Tobruk, and Burma, contracted septic infections, epidemic jaundice, dysentery, scrub typhus, and malaria, and fell a victim to diabetes. In establishing a claim for pension it was not a question of war conditions only but war conditions plus repeated infections to which this man would not have been otherwise subjected and which were likely to antedate the onset of diabetes in a predisposed individual.

Where the onset of diabetic symptoms follows immediately upon an incident under Service conditions—e.g., a head injury in a motor-cycle accident or on immersion in the icy North Sea, both examples of actual cases—no one can maintain that the accident had nothing to do with the onset of diabetes. In fact any unprejudiced observer, be he medical, scientific, or lay, would take the view that cause and effect might reasonably be associated here. Furthermore, as I understand the position, it is not for the claimant to prove cause and effect without the shadow of a doubt, but for the Ministry to show without any doubt that the incident and the onset of diabetes could not have been in any way associated. I should therefore still

make the plea that where there is any possibility that diabetes occurred as a result of accidents or strain during service the case of the claimant should get a fair and full hearing.

I cannot agree with Dr. Lawrence that diabetes is in every instance a disease which can be so lightly regarded that it will not produce disability and some incapacity in earning a living. The severe diabetic is liable to sepsis following minor injuries and abrasions and to major effects from minor infections. He may be unsafe in occupations involving proximity to dangerous machinery, and in particular he may easily fall a victim to tuberculosis. I have already seen this occur in a diabetic pensioner. In conclusion, I still feel very strongly that the diabetic is worthy of his pension.—I am, etc.,

Aberdeen. A. LYALL.

Bornholm Disease

SIR,—Dr. A. F. T. Ord's admirable description (Nov. 20, p. 916) of his cases comes close to Daa's original description of the syndrome (as quoted by Sylvest[1]) which he named epidemic myalgia: "As a rule the patient has a stitch in one side of the chest . . . which is often accompanied by pains in the back, shoulders, epigastrium, and abdomen." Several outbreaks of epidemic myalgia have been described in the literature,[2 3] and most observers agree that there are no *x*-ray changes demonstrable in the disease. In this connexion it is interesting to note that if the film was correctly interpreted some mid-zone opacity was seen in Dr. Ord's first patient's chest, thus bringing atypical pneumonia into the differential diagnosis.

The other syndrome which ought to be considered in the differential diagnosis is the non-glandular form of infectious mononucleosis. In a recent paper[4] I have described the extremely difficult differential diagnosis between the atypical forms of infective mononucleosis, epidemic myalgia, and the pre-paralytic stage of acute poliomyelitis—an important point in times of an epidemic.

I cannot but concur with Dr. Ord's criticism of the indiscriminate use of the "sulpha" preparations and penicillin in these cases. Some yet unpublished therapeutic response has been demonstrated in at least infectious mononucleosis with para-aminobenzoic acid (P.A.B.A.). It is possible that the drug may be effective also in the more uncommon epidemic myalgia, and it is obvious that to give these patients sulphonamides is just the wrong thing to do.

The differential diagnosis can be made easier if white counts are taken of these patients, a procedure certainly easy for a hospital and accessible even to a general clinician. In a case of frank pneumonia warranting chemotherapy or penicillin one would expect marked leucocytosis with increased polymorphonuclears, except in a very few toxic cases of bone-marrow depression. The white counts in epidemic myalgia are either normal or only very slightly increased, and in infectious mononucleosis the diagnosis can be safely made by demonstrating the typical young mononuclears ("atypical lymphocytes" of American writers). As a matter of fact the differential white count is the only safe way of diagnosis for the latter syndrome, since the Paul–Bunnell reaction is positive only in less than 50% of the cases during the first fortnight, and cannot be demonstrated at all in almost 30% of the cases.[5]

To sum it up, a little more clinical observation, like that demonstrated by Dr. Ord, and much less mechanized medicine, and the list of "rare" and "uncommon" diseases will have to be radically revised.—I am, etc.,

London, S.W.7. A. ERDEI.

REFERENCES

1 *Epidemic Myalgia*, 1934, London.
2 Nichamin, S. J., *J. Amer. med. Ass.*, 1945, **129**, 600.
3 Scadding, J. G., *Lancet*, 1946, **1**, 763.
4 *Canad. med. Ass. J.*, 1948, **59**, 159.
5 Press, J. H., Shlevin, E. L., and Rosen, A. P., *Ann. intern. Med.*, 1944, **22**, 546.

Out-patient Electric Convulsion Treatment

SIR,—Dr. J. F. Cooper's letter (Nov. 13, p. 878) prompts me to report that at the Middlewood out-patient clinic E.C.T. has been used regularly since 1941, with results very similar to those recorded in Sir W. P. Mallinson's article (Oct. 2, p. 641). We can confirm the safety of this form of treatment when carried out by experienced psychiatrists with facilities for the immediate admission of the patient when desirable. In over 3,000 treatments no complications, fractures, or untoward incidents have occurred. No restraint has been used apart from a loose drawsheet passed around the trunk and arms. Oxygen has never been used, and complaints of post-convulsive headache have been few and hardly worth mentioning. Three nurses are utilized, mainly for the purpose of transport of patient to the recovery rooms and subsequent supervision.

Fear of treatment is a noticeable feature in a minority of patients, and probably the majority would confess to some apprehension if asked about it, but the benefit derived from treatment far outweighs these misgivings. The number of treatments given in one course seldom exceeded eight, and I can recall only one or two patients in each year who gave up attending through fear. For the latter patients and for those who experience a post-convulsive period of excitement Dr. Cooper's recommendation of premedication with small doses of pentothal is of value, but I doubt the wisdom of complicating the procedure by routine intravenous injections.—I am, etc.,

Middlewood Hospital, Sheffield. F. T. THORPE, Medical Superintendent.

Clouding of Surgeons' Spectacles

SIR,—Like most surgeons who wear glasses, and particularly those who prefer a mask with a cellophane layer, I have tried many methods of preventing steaming. Until we get our "green forms" for the special glasses recently described, may I recommend the following simple method which was evolved under the testing conditions of a hospital where the atmosphere was particularly humid as well as very hot?

A thin piece of soft cotton-wool about 5″ × 2″ is partly torn across the middle to give it a butterfly shape; the "butterfly" so formed is placed along the upper edge of the mask before it is assumed. It causes no irritation, and experiment soon determines the smallest amount of wool which will prevent one's breath from passing between the mask and the nose. Unlike special preparations for the glass, which soon lose their effect, the wool enables one to pass the longest, hottest morning's operating with unclouded glasses.—I am, etc.,

Birmingham. G. K. ROSE.

Psychiatry

SIR,—Whilst I am grateful to your critic, Dr. Eliot Slater (Nov. 20, p. 906), for reviewing my book on psychiatry, I feel, nevertheless, obliged to register a mild protest against some of his remarks. A book which sets out to deal with the whole of the subject of psychiatry in some 380 pages cannot, surely, afford to be "full of asides." The latter were intended to be not off-stage whispers but positive and fully thought-out statements and reflections, whose extra-textual quotation can only be calculated to distort their meaning.

The impression given by Dr. Slater that the book emphasizes body–mind dualism overmuch must, I fear, be imputed to his reading of it, not to my writing. Indeed, the theory embodied in the book is in effect a mind–body monism—if I may so put it. It purports, furthermore, to treat psychiatry as if it were concerned with mental illness, with psychic deviations from an accepted norm. If in championing this viewpoint I have run counter to the, at present, more fashionable notion of looking at the subject from a physical angle, it is regrettable that I should be chastised for it.

Dr. Slater quotes me as saying, or rather meaning, that "preclinical disease is an unconscious process." Yes, indeed it is. A conglomerate of carcinomatous cells, for instance, may lead its parasitic existence for quite a time to the complete unawareness of its host—would that it were otherwise! But I went much further than this. I meant to convey the assumption that unconscious dynamic processes and physical disease might well stand towards each other in the relationship of cause and effect, or at the very least as concomitants in one single and indivisible process.

I am well acquainted with Dr. Eliot Slater's learned work on genetics, as well as with his orientation in psychiatry, and I have met him on several occasions. May I end by thanking him for not sparing the rod, and for wielding it with more grace and charm than is usual among psychiatric opponents?—I am, etc.,

Kingswinford, Staffs. W. A. O'CONNOR.

"Dye"

SIR,—I note a regrettable tendency on the part of radiologists and other clinicians to refer to many contrast media used in radiology as "the dye." The *O.E.D.* gives the following meanings of the noun: (1) colour produced by dyeing, and (2) material used for dyeing.

Is it really necessary to be so slipshod in the use of our native tongue, even for the sake of brevity? The fact that tetraiodophenolphthalein is, until acidified, a blue dye is no justification for so describing such colourless substances as 'pheniodol," iodoxyl, diodone, "thorotrast," and iodized oil. If this goes on we shall soon be giving barium dye to show a duodenal ulcer. I write in the hope that this habit will soon die a natural death.—I am, etc.,

London, W.1. S. COCHRANE SHANKS.

Eye Bank Needed

SIR,—I am an Australian and have just returned to London after several months of study in the U.S.A. I feel there is a very real need in England for the establishment of an eye bank. The American eye bank is very busy and functioning splendidly. It has enabled thousands of corneal grafts to be done which have undoubtedly restored vision to many blind people. It receives as much support from the American people as the blood bank. In a small country like Britain an eye bank should be very easy to organize.—I am, etc.,

London, W.C.2. F. W. SIMPSON.

Marxist Genetics

SIR,—Dr. C. D. Darlington's sarcasm at the expense of *Soviet Biology* (Nov. 13, p. 862) might have been more effective if it had been more accurate. A particular example of this inaccuracy is his reference to "the idealistic metaphysical bodies the chromosomes," implying that this is Lysenko's attitude. In fact Lysenko states, "We recognize the chromosomes, we do not deny their presence. . . . Is heredity transmitted through the chromosomes in the sexual process? Of course it is" (p. 43). Again, Dr. Darlington remarks on the alleged deletion from Lysenko's report of any reference to his views concerning the competition of members of the same species. Yet if only Dr. Darlington had read as far as p. 38 he would have seen an explicit reference to this very theory.

Dr. Darlington also refers to what he terms "a new figure in Soviet mythology" and adds, "We shall all watch Williams." I wish him well in his watching, for Williams died 20 years ago. He was a very well-known Russian soil biologist whose ancestors emigrated there. A review containing such inaccuracies as this cannot be taken seriously. Dr. Darlington appears to have been chiefly motivated by reasons other than scientific. Lysenko's report, however, is well worth serious study to all those genuinely interested in genetics. He is by no means an unscientific farmer, but a scientist well conversant with the current theories of formal genetics.—I am, etc.,

London, N.W.3. BARBARA MCPHERSON.

Safer Milk

SIR,—In the annotation entitled "Safer Milk to Come" (Nov. 13, p. 868) you say, "About one quarter of the milk supplied to schools is neither from a T.T. herd nor has it been pasteurized." This is incorrect. Figures for school milk at the beginning of this year were:

England and Wales:	
Pasteurized	92 %
T.T.	4.6%
	96.6%
Scotland:	
Pasteurized	52.2%
T.T.	46.8%
	99.0%

The few schools which still receive ordinary raw milk are in remote places, such as in the Welsh mountains, where no pasteurized or T.T. milk is available but where the medical officer of health has approved a local supply which is kept under close supervision.—I am, etc.,

London, W.1. W. A. LETHEM, Ministry of Food.

POINTS FROM LETTERS

Breast-feeding

Dr. CHARLES A. MARSH (Bath) writes: Another cause of the failure of lactation is the abolition of the free movement of the baby's arms. In normal lactation the baby's hands gently massage the mother's breast, and this is a most important factor in the flow of milk. The modern plan of wrapping the baby up so completely that the arms are invisible and useless, thus turning it into a mummy, is altogether contrary to nature and limits the co-operation between mother and baby so well described by Dr. Rachel Pinney (Nov. 13, p. 878) as a love act. A calf or a lamb constantly varies suction with bumping its head against its mother's udder; massage by the baby's hands is just as natural though less violent. The only reason I have been given for wrapping the arms is to prevent the baby scratching its face, involving possible criticism of the nurse, but this can be met by cutting the baby's nails. The fact that there is little difference in the amount of breast-feeding in hospital or at home does not affect this question, as the district midwives get the same training. The old-fashioned tight binder has gone out and the new-fashioned mummification come in. I do not know which of the two is worse.

Dr. C. T. NORRIS (London, S.W.6) writes: With experience similar to Dr. James S. Hall (Nov. 20, p. 919) I have only found one mother among all those *who really want to feed their babies* who could not do so when afforded adequate supplies of calcium and iron, which I now use in the form of calcium lactate gr. 10 or 15 (0.65–1 g.) in 2 dr. (7 ml.) of the *N.W.F.* mistura ferri et ammonii citratis, t.d.s. The criterion is not quantity only; a thin watery milk, however plentiful, requires this treatment, and so does a mother, whatever the lacteal condition, who resumes the puerperal flow on returning home or who menstruates during lactation. These facts are not and cannot be known to those who lose sight of the mother 10 or 14 days after delivery, in whose hands most of the midwifery now lies, and who consistently fail to induce the mother to see her own doctor for further advice when they send her out of their care. They also do harm by sometimes advising 3-hourly feeds, against which no mammary organization I have met can contend indefinitely. . . . In at least half the cases where the baby is presented as the patient for a vague asthenia or wind, treatment is needed on these lines for, and through, the mother.

Carcinoma of Cervix

Dr. E. MALCOLM CLARK (Fort Hall, Kenya) writes: Mr. Sampson Handley has stated (Dec. 20, 1947, p. 1010) that the relative freedom of Jewish women from carcinoma of the cervix was almost certainly due to circumcision of Jewish males. He also quotes the case of circumcised Fijians, and even goes so far as to state that as a consequence of present standards not being as far ahead as the hygiene code of Moses thousands of women are annually sacrificed to this form of cancer. Amongst the Kikuyu tribe in Kenya all the males are without exception circumcised before puberty. Carcinoma of the cervix amongst the females of the tribe is, however, so far from being rare, the commonest of all forms of cancer. In a series of 82 consecutive cases of cancer amongst the Kikuyu (both sexes) I found no less than 12 cases of carcinoma of the cervix. With one exception the diagnosis was confirmed by histological examination at the Medical Research Laboratory, Nairobi. One case not so confirmed was operated on by a very experienced gynaecologist in Nairobi. It was found that owing to the advanced state of the growth it was impossible to perform a hysterectomy; there could be no possible doubt as to the diagnosis. It would not, I think, be justifiable to attribute the low incidence of carcinoma in Jewish and Fijian women to circumcision in their men-folk. Rather would it not be safer to suggest some racial factor which we do not as yet fully understand?

Another Unusual Case of Twins

Dr. J. K. POMEROY (Lemberg, Canada) writes: With reference to the memorandum on an unusual case of twins by Drs. J. H. Young and F. E. Cull (Oct. 16, p. 713), I was called to a maternity case thirty miles away on April 5, 1948. When I arrived the woman was in labour. I diagnosed twins. I had not been in the house many minutes when I delivered the first foetus, a girl, by the breech and divided the cord. The next presenting object was a placenta. At this point there were two cords leading into the uterus and at the external end of one a placenta was attached. Then came the second foetus, a boy, vertex presenting. As I had not severed the cord, there I was with the second foetus connected by an intact cord with its placenta—i.e., the placenta that had already been expressed. The placenta belonging to the first foetus was expressed in a few minutes. The woman made an uneventful recovery. I do not know the woman's history, as I saw her only the once. I do know she was a bipara, with the first child about 4 years of age. It is a rare condition. The sequence was the same as Young and Cull's, foetus A, placenta B, foetus B, placenta A.

Obituary

STUART McDONALD, M.D., F.R.C.P.Ed.

Many of those who were students in medicine at Newcastle-upon-Tyne before the last war and many of the older members of the Pathological Society will learn with regret of the death on Nov. 15 of Stuart McDonald, emeritus professor of pathology in the University of Durham.

Stuart McDonald was born at Castle Douglas and educated at Dumfries Academy, going on to the University of Edinburgh, where he graduated M.B., C.M. in 1896. In 1907 he proceeded M.D., obtaining a gold medal for his thesis on subacute necrosis of the liver, a subject of which much less was known then than is the case to-day. Shortly after qualifying he began to specialize in pathology, spending some time at Freiburg, where he learnt much from the teaching of Aschoff. Then for some years he lectured on his specialty in the extramural school at Edinburgh. In 1909 McDonald was appointed to the chair of pathology in the University of Durham, and there he remained for the next twenty-eight years, till his retirement in 1937. He published little in his later years with the exception of an important study on asbestosis in 1927, but he was a constant attendant and a well-known figure at the bi-annual meetings of the Pathological Society. He threw himself with zest into the life of the University, becoming dean of the medical faculty and an officer in the Medical Detachment of the Officers' Training Corps as well as taking a keen and active part in many student activities. He was also deeply interested in forensic medicine and became known throughout the North of England as a reliable expert witness. Professor McDonald was for long a member of the Newcastle-upon-Tyne and Northern Counties Medical Society and eventually became its president. His services were much in request as an external examiner in pathology in many universities in England and Scotland, and in addition he served both as secretary and as vice-president of the section of pathology at B.M.A. Annual Meetings.

Despite his love of cities McDonald was essentially a countryman who looked forward with zest to the days that he could snatch beside his beloved trout streams. His years of retirement were spent among the quiet Peeblesshire hills, where he could indulge to the full his taste for rural things. His life at the end was saddened by the death in 1946 of his son, who was professor of pathology in the University of St. Andrews.

J. B. SIMPSON, C.B.E., M.D., F.R.C.P.Ed.

Dr. James Bertie Simpson, of Golspie, died on Nov. 16 in Edinburgh at the age of 85. Dr. Simpson was a student at Edinburgh University, and graduated M.B., C.M. in 1887, proceeding M.D. in 1892 after a period of study at Leipzig and Vienna. He was elected F.R.C.P.Ed. in 1925, and was a deputy-lieutenant for the county of Sutherland. He was awarded the O.B.E. in 1920 and was made C.B.E. in 1942. Dr. Simpson was an assistant in Ballachulish, Argyle, before settling in Golspie. He was chairman of the Caithness and Sutherland Division in 1922–3 and again in 1941. He had been a member of the Highlands and Islands Consultative Council and of the Insurance Acts Subcommittee for Scotland. He was an active member of the Scottish Committee from 1933–40 and again in 1945–6. He was also a representative at Annual Representative Meetings on several occasions.

Dr. D. W. D. MacLaren writes: With the passing of Dr. J. B. Simpson our profession in the North of Scotland mourns the loss of its most honoured and distinguished representative. "Simpson of Golspie" was indeed a household word in all our northern counties. His devoted work over many years on behalf of his medical colleagues can never be forgotten. He was a keen and enthusiastic "B.M.A. man," holding the highest positions in his Division and Branch, and for years the trusted delegate to its national councils and committees in Edinburgh. His dynamic and colourful personality found its scope in so many local and county activities that it was always a source of amazement how he found time for them all. In addition he ran a large and most successful general practice, and was medical superintendent of the county hospital at Golspie. He had the highest ideals and principles, and he was always a wise guide and friend to all who sought his advice. With his natural gifts of mind and heart he brought exceptional skill and competence to his professional work in Sutherland, that county which he served so well and which he loved so much. We shall always recall the energetic figure, the fresh complexion, the twinkling eye, the ready wit—caustic at times, but never vindictive—and the abounding humour, wise judgment, and shrewd Scottish common sense of this remarkable doctor, sportsman, and friend. To his son, Mr. B. Soutar Simpson, consulting surgeon to the county of Sutherland, who continues the family tradition, and to all those near and dear to him we offer heartfelt sympathy.

Dr. R. W. Craig writes: With a cultured mind and a capacity for shrewd judgment Dr. J. B. Simpson combined a rich sense of humour and a strong appreciation of the finer things of life. Straight as a die, he could not tolerate humbug in any shape or form. His penetrating eye, despite its twinkle, could exert an influence which was frequently both chastening and astonishing. As a raconteur he was inimitable, and to spend an evening in Simpson's company was a most refreshing and delightful experience. To the British Medical Association he gave ungrudging and loyal service, but it was only natural that his chief interest should lie in the work of the Highlands and Islands Subcommittee, of which he was chairman for many years. It was in large measure due to his influence that the Highlands and Islands Medical Service achieved its world-wide reputation. In the home of the crofter or the laird he was equally beloved and respected. He was a keen angler and an expert deerstalker. He kept himself fully abreast of the latest developments in medical science, and this is reflected in the fact that the late Sir John Fraser was glad on occasion to give his class of clinical surgery the opportunity of having a lecture from Dr. Simpson. A fine example of the best type of family doctor, his character and work will remain an inspiration to all who had the privilege of knowing him.

Dr. GEORGE JAMES IRVINE LINKLATER, chief executive school medical officer to Edinburgh Corporation, died in the City Hospital in Edinburgh on Nov. 24 at the age of 57 after an illness of some months' duration. Dr. Linklater had been in the service of the Edinburgh Corporation for twenty-six years. He graduated at Edinburgh in 1912 and proceeded M.D. in 1920. Over the next two years he took the M.R.C.P.Ed. and the diplomas in public health and in tropical medicine. Dr. Linklater was a keen Territorial and served in the R.A.M.C. in both world wars. He received the O.B.E. for his services in the 1914–18 war, and in the recent war he held the rank of colonel; for his services to the Red Cross he was awarded the Order of St. John. He had also been for many years a High Constable of Holyroodhouse. Before his appointment as school medical officer in 1930 Dr. Linklater had been a resident physician in the City Fever Hospital and in the Royal Victoria Hospital, and from there he went on to become a tuberculosis officer in the city. With Dr. John Guy, a former medical officer of health, he was the joint author of a book, *Hygiene for Nurses*, which has been the stand-by of many nurses for years. Dr. Linklater was one of Edinburgh's ablest administrators, and the present high standard of the school medical service is due to his unceasing work. He was at his best with children, as those who saw him hold the rapt attention of large audiences at the junior cinema clubs on Saturday mornings would know. He was an able and lucid lecturer and took a leading part in teaching hygiene to nurses and health visitors. A delightful raconteur and a good companion, he will be sorely missed by a wide circle of friends.—W. G. C.

Dr. L. Carlyle Lyon writes: It is with profound regret that I, one of his grateful Aberdeen students in the years 1922 to 1927, learned of the death of Professor Theodore Shennan (*Journal*, Nov. 6, p. 839). His value as a teacher was due in part to the unusual personal interest he showed in each one of the students in his class, even to the point of obtaining their individual photographs. His friendliness created enthusiasm for his subject and transformed hard work into a human relationship. He was invariably at his best in the post-mortem room, and his textbook remains, in my opinion, a fitting record of his thorough mastery of his subject. All those who studied under him will feel a very personal loss.

Medical Notes in Parliament

Sanitary Conditions in the Catering Trade

Mr. EDWARD DAVIES on Nov. 18 drew attention to the use of cracked and chipped crockery, particularly in catering establishments, and the relation of this use to the spread of infection and disease. Dr. BARNETT STROSS said that from what little bacteriological work had been carried out it appeared that reasonable cleansing of an unbroken service with boiled water would leave only a few organisms, whereas on cracked crockery there could be found many types of organism, particularly haemolytic streptococci. Medical students used to hear terrifying stories about diseases which could infect the lips from the use of dirty cups by infected people. They now knew that this happened very rarely, but trench mouth had been prevalent during the war and there was some cause for thinking that it could arise from cracks in pottery.

Dr. EDITH SUMMERSKILL said that the Medical Research Council had experimented on china in various stages of dilapidation, and the viable bacterial count on the mouth area of a cup was found to vary from a few organisms to many thousands. The number of these organisms was not determined by the cracks or chips but by lack of cleanliness. The Ministry of Health had no record of any serious illness that had been traced to the use of cracked crockery. Apart from sore throats, which could be attributed to streptococci, there was no trace of any other outbreak. Though germs might be left in cracks after the use of boiling water, there must be a heavy dose of virulent streptococci before anyone could be infected. Cases of food-poisoning which had been reported all over the country were not attributable to infected cups, spoons, or forks but to food in which organisms had multiplied rapidly. It was quite impossible for organisms to multiply rapidly in cracked cups. Whatever the material used to make utensils, the only safeguard was to ensure that they were properly cleaned. In restaurants throughout the country these utensils were rinsed in water which teemed with bacteria and were wiped with cloths containing many thousands of living bacteria to the square inch. The Ministry of Health was trying to educate the public on the need for a better service. She thought it would be possible for the large catering establishments to give a preliminary wash in a first sink of water containing a detergent at a temperature of 110 to 115° F. (43.3°–46.1° C.) followed by a rinse in a second sink of water at a temperature of about 175° F. (79.5° C.) for at least thirty seconds, the articles then being allowed to drain dry without wiping. That was much better than wiping them with a filthy cloth covered with bacteria. Crockery so treated, though not sterile, would have a low bacterial count.

Dr. Summerskill reminded the House that at present any individual could inform upon the manager of a catering establishment who conducted it in a dirty fashion. He could ask the local authority for an inspector to inspect the establishment. The Ministry felt so seriously about this matter that it was setting up a catering trade working party with the following terms of reference: "To make recommendations to the Ministers of Food and Health and the Secretary of State for Scotland as to the precautions considered practicable and desirable with a view to securing the observance of sanitary and cleanly conditions in the catering trade."

The chairman of this working party would be Sir William Savage, an authority on food hygiene. It also included a medical officer of health, a sanitary inspector, Dr. Sutherland, of the Central Council for Health Education, medical and administrative officers from the health departments at the Ministry of Food, and persons drawn from different sections of the catering trade. This working party would hold its first meeting on Nov. 26. Dr. Summerskill added that supplies of pottery were improving, and she learned from the Board of Trade that caterers were now taking more than 10% of the supplies available in the home market. It was essential, however, to increase exports, and therefore it was impossible for Mr. Strachey to make a statutory provision prohibiting the use of chipped crockery.

Civil Defence: Medical Arrangements

Speaking on Nov. 23 at the close of the debate on the Second Reading of the Civil Defence Bill, Mr. ANEURIN BEVAN said it was not necessary to have an emergency medical service to-day, "because the health and hospital services of the country are already instruments of the Government." There had not got to be elaborate co-operation with voluntary hospitals. Arrangements had not to be made with local authority hospitals. Hospitals were now organized on a national scale. It was now only necessary for the Minister of Health to ask regional hospital boards to identify sites and buildings where they could expand hospital services in an emergency. The same thing was true of blood banks. Blood transfusion services were nationally organized and nationally administered. The same was also true of the ambulance services, now the responsibility of the counties and county boroughs and capable of being expanded at any time. So the Government by its wisdom in constructing the National Health Service had at the same time provided the foundations for an effective defence organization.

Tuberculous Ex-Servicemen in Switzerland

Mr. CHETWYND on Nov. 23 asked whether the Minister of Pensions had any statement to make on the results of the scheme initiated by the Don Suisse for the treatment in Switzerland of ex-Servicemen disabled by tuberculosis.

Mr. H. A. MARQUAND said he would circulate in the official report a short account of the scheme under which, through the generosity of the Don Suisse organization, 180 British ex-Service and Service men suffering from pulmonary tuberculosis were provided with free sanatorium treatment in Switzerland. He added that he had conveyed to the Swiss Minister the cordial thanks of His Majesty's Government for the generous and friendly gesture of the Swiss people.

The account which Mr. Marquand supplied indicated that in 1946 the Don Suisse offered to provide, for one year, 100 beds in sanatoria at Leysin, in Switzerland, for the free accommodation and treatment of British ex-Servicemen suffering from pulmonary tuberculosis. It was arranged that the patients should be selected by the Ministry of Pensions according to clinical criteria agreed with the Swiss doctors. Medical officers of local health authorities were consulted, and patients were selected as follows: from Service hospitals 28; from local authority sanatoria: English counties and county boroughs 87, London County Council 34, Scotland 21, and Wales 10. The first hundred patients went to Switzerland in January–February, 1947, in six parties. In August, 1947, 76 patients returned on completion of the agreed period of treatment and 79 others went out. Seven patients returned in December, 1947, and 37 in April, 1948. The remainder came back in small groups each month up to Aug. 31, on which day the scheme ended, having been extended by eight months through the generosity of Don Suisse.

Basic Salary

Dr. SEGAL asked on Nov. 25 by what authority local executive councils were empowered to ask a doctor applying for payment of basic salary to submit details of his income from all sources. He demanded an assurance that a doctor who refused to divulge these details would be in no way prejudiced when his claim for a basic salary came up for consideration.

Mr. BEVAN said his view was that it was only the doctor's professional income which should be taken into account in such cases.

Dr. SEGAL asked whether it would be advisable to investigate incomes at the other end of the scale where overworked doctors might have lists of patients with which they could not cope and might be receiving incomes larger than they ever had before.

Mr. BEVAN said that was entirely another question. That matter was being reviewed by the Medical Practices Committee. Until the Ministry had received a survey of the whole country it would not be able to identify those parts where the lists were too large and the areas under-doctored.

Mr. RANKIN said that doctors who applied for the basic salary were told that that salary, if paid, was a charge on their colleagues in the area in which they practised.

Mr. BEVAN replied that it was true that the £300 a year came out of the general capitation pool, as indeed it ought to do because it formed part of a doctor's remuneration. Where, however, an individual doctor was aggrieved, he had the right of appeal to the Minister. Many had so appealed, and decisions had been given.

Distribution of Doctors

Mr. BOSSOM asked how many more doctors were required to enable the service promised under the New National Health Scheme to be given satisfactorily, and what special efforts were being made to train doctors to supply this need.

Mr. BEVAN said distribution was as important as total numbers. The Medical Practices Committee was obtaining reports from all areas in England and Wales as at Dec. 31 and would then be able to assess where the main under-doctored areas were. The medical schools were already filled to capacity.

Mr. BOSSOM asserted that there was a shortage of about 20% of the doctors required.

Mr. BEVAN said this was quite incorrect. He added that until the Medical Practices Committee reported he was not in

a position to say what areas were under-doctored. He had no information that the medical profession itself said that approximately 20% were required. It was, however, a fact, he said, that doctors in particular areas were not anxious to say their areas were under-doctored.

Mrs. MANNING asked Mr. Bevan to consult Sir Stafford Cripps about expanding the medical schools at a very early date.

Mr. BEVAN answered that these schools were filled to capacity. It was impossible to expand them beyond the capacity of teachers available to teach in them. The medical teaching staffs formed the limitation.

Colonel STODDART-SCOTT asked whether Mr. Bevan was prepared to carry out the recommendation of the Goodenough Committee to start another medical school.

Mr. BEVAN replied that many recommendations of the Goodenough Committee were already in operation, but it was useless to open medical schools if they did not have staff for them.

Prescribing for School-children

Mr. YORK on Nov. 25 asked whether Mr. Bevan would provide for the supply, free of charge, of medicines and drugs prescribed by school medical officers at school clinics.

Mr. BEVAN answered that medicines supplied at a school clinic would be supplied without charge. If the school medical officer thought a pupil required other medicines he could refer the patient to his National Health Service doctor, who could prescribe for free supply under the Service.

Medical Practices Committee.—Mr. BEVAN informed Sir Henry Morris-Jones on Nov. 15 that up to that date he had received no recommendations from the Central Medical Practices Committee on the question of maldistribution of practitioners. That Committee would obtain, by Dec. 31, comprehensive reports on the adequacy of the medical services throughout England and Wales. In the light of these reports he would carefully consider any recommendations which the Committee saw fit to make to him.

B.C.G.—On Nov. 18 Mr. WARBEY asked the Minister of Health what arrangements were being made for a trial of B.C.G. vaccine in this country, with a view to better protection of nurses, doctors, and other contacts? Mr. BEVAN answered that arrangements were nearing completion. He was not yet in a position to make any useful public statement on them, but would do so as soon as possible.

Hospital Charges.—Mr. PETER FREEMAN asked Mr. Bevan on Nov. 18 what charges, and under what conditions, hospitals controlled by his department could impose on patients; and whether they were informed on admission of such charges. Mr. BEVAN replied that patients could go into pay-beds, at full cost. Otherwise they could only be charged for extra privacy in single rooms or small wards, for the extra cost of certain expensive appliances, and for renewal of appliances damaged through negligence. They were so informed on admission.

Venereal Diseases.—The National Health Service (Venereal Diseases) Regulations, 1948, were presented to Parliament on Nov. 22.

Milk for Old People.—Dr. SUMMERSKILL said on Nov. 22 that milk supplies were not yet sufficient to allow extra milk to old people. There were 3,000,000 persons in the United Kingdom above the age of 70 years.

Equal Pay for Women.—Mr. BEVAN announced on Nov. 25 that the differences in the scales of pay between men and women administrative officers in the National Health Service were being abolished.

X-ray Equipment.—Hospitals in the National Health Service have received 179 diagnostic *x*-ray units under the arrangements for central provision. Some types can now be supplied from stock. All hospitals have been asked to state their needs.

Waiting List.—Persons suffering from tuberculosis who awaited admission to hospital on June 30, 1948, numbered 9,208.

Jamaica.—Of the total births registered in Jamaica in 1946, 67.71% were illegitimate. There are at present 5,576 free hospital beds in Jamaica to meet an estimated need of 7,850.

Universities and Colleges

ROYAL FACULTY OF PHYSICIANS AND SURGEONS OF GLASGOW

Professor James C. Spence will deliver the Finlayson Memorial Lecture in the Hall of the Faculty (242, St. Vincent Street, Glasgow) on Wednesday, Dec. 8, at 5 p.m. His subject is "Poliomyelitis." Medical practitioners are invited to attend the lecture.

Medical News

International Congress of Ophthalmology

Their Majesties the King and Queen have been graciously pleased to grant their patronage to the sixteenth International Congress of Ophthalmology, which will be held in London under the presidency of Sir Stewart Duke-Elder from July 17 to 21, 1950. Any qualified medical practitioner may become a member of the Congress; others not so qualified who have rendered service to ophthalmology and who are approved by the Council will be eligible to attend. Relatives and friends of members will be welcomed as associate members and may attend the opening and closing ceremonies and all social functions. The official languages are English, French, and Spanish: any language, however, may be used. The International Council of Ophthalmology has agreed upon two main discussions on the following subjects: (*a*) the role of the sympathetic system in the genesis of vascular hypertension and its effect upon the eye; (*b*) the clinical and social aspects of heredity in ophthalmology. Those desirous of participating in the main discussions or reading communications on other ophthalmological subjects should communicate with Mr. Keith Lyle, International Congress of Ophthalmology, 45, Lincoln's Inn Fields, London, W.C.2.

Malta Hospital Rebuilt

The King George V Merchant Seamen's Memorial Hospital in Malta has been rebuilt and was opened by Lady Mountbatten on Nov. 30. The hospital was originally opened in 1922 and was intended chiefly to serve the needs of merchant seamen. In addition, however, it has always cared for the personnel of the Royal Navy and their families, and residents and visitors to the Island. It was destroyed by enemy action on April 7, 1942. The Scottish Branch of the British Red Cross Society contributed £125,000 to rebuilding the hospital, and other organizations and friends have contributed large sums.

Historic Medical Diary

On display in the rare manuscripts room at the British Museum are the diaries and papers from Scott's last expedition. An interesting item is the 1910 "Wellcome" Medical Diary carried throughout his journey by Dr. E. A. Wilson, medical officer to the party. As legible as if it had been written to-day, it forms a record of personal and scientific observation second in importance only to Captain Scott's own papers. The diary was found in the tent where the South Polar explorers met their end.

Radioactive Tracers for Germany

A contract for the supply of radioactive tracers to approved research institutes in the Bizone has recently been placed with the Atomic Energy Research Establishment, Harwell, by the Joint Export Import Agency. This follows the decision of the Isotope Allocation Committee of the Ministry of Supply in January last that radioactive tracers produced in the Harwell "gleep" could be made available for research work in Germany. The decision of the Isotope Allocation Committee was communicated to the authority responsible for the control of research in the British Zone, who immediately informed the U.S. Research Control Group. Together they approached the Board of J.E.I.A., supported by their respective public health advisers; and with the minimum of delay the Board authorized the import of radioactive tracers from Harwell into the Bizone. Research institutes in the Bizone thus gain access to a range of radioactive tracers the indigenous production of which is denied to Germany under Control Council Law No. 25 for the Control of Research. Their allocation within the Bizone is the responsibility of a small German committee competent to decide where proper use will be made of the small total supply of tracers at present available. In order to assist the committee in its task a team of experts from A.E.R.E. visited Germany during October, demonstrated the latest ancillary equipment, and answered many queries put to them by the committee.

Admission to Roll of Assistant Nurses

The General Nursing Council for England and Wales announces that people wishing to apply as assistant nurses with intermediate qualifications for admission to the Roll of Assistant Nurses set up under the Nurses Act, 1943, must do so before Jan. 1, 1949. After that date only those people who have undergone two years' training as pupil assistant nurses in a training school approved by the council and have passed a test laid down by the council will be admitted to the Roll. Under the Waiver Rule applicants who would have been eligible for enrolment as existing assistant nurses, but who failed to apply before Feb. 3, 1946, may now be considered for enrolment. Information may be obtained from the Registrar, General Nursing Council for England and Wales, 23, Portland Place, London, W.1.

Marriage Guidance

The "Report of the Departmental Committee on Grants for the Development of Marriage Guidance" has just been published. The Committee, which was set up in February of this year by Mr. Chuter Ede, recommends that the work in marriage guidance of the National Marriage Guidance Council, the Catholic Marriage Advisory Council, and the Family Welfare Association should be given help from public funds. It is suggested that these three bodies should together devise schemes for selecting and training persons who are acting as marriage counsellors. It is proposed also that ministers of religion might be allowed to take advantage of these schemes of training. The three organizations are not to lose their identities, and Government grants are recommended of £5,000 a year for the National Marriage Guidance Council and £1,500 a year for the Family Welfare Association and the Catholic Marriage Advisory Council. The Committee proposes that the grants should be reviewed from year to year over an experimental period. The setting up of local centres for marriage guidance is recommended, and it is proposed that such centres should be supported financially by the local authority, one half of any substantial expenditure incurred by the local authority being met by a Government grant.

Dr. E. H. Taylor-Cummings

Dr. E. H. Taylor-Cummings, M.B.E., J.P., was elected Mayor and Alderman of the Municipality of Freetown, Sierra Leone, on Nov. 16.

Dr. G. M. Findlay

Dr. G. M. Findlay, editor of the B.M.A. Abstracting Service, has been elected a Corresponding Member of the Société de Pathologie Exotique.

Sir Arnold Stott

Sir Arnold Walmsley Stott, K.B.E., B.Ch., F.R.C.P., has been appointed an Extra Physician to the King's Household.

New Sheriffs

David Rhys Lewis, M.D., F.R.C.P.Ed., has been nominated a Sheriff in the County of Breconshire, and Dyfrig Huws Pennant, D.S.O., M.D., in the County of Pembrokeshire.

Director-General of Unesco

Señor Torres Bodet, Foreign Minister of Mexico, has been elected Director-General of Unesco in succession to Dr. Julian Huxley. Señor Bodet was the only candidate.

COMING EVENTS

South-West London Medical Society

The third clinical meeting of the session will be held at the Bolingbroke Hospital at 8.15 p.m. on Wednesday, Dec. 8, when Mr. Wm. A. Hervey will lecture on "Fenestration—Hearing Aids."

Public Health Centenary

The centenary of the Public Health Act, 1848, will be celebrated by the Trustees of the Chadwick Trust at the Royal Society of Tropical Medicine and Hygiene (26, Portland Place, London, W.) on Tuesday, Dec. 14, when tea and cocktails will be served between 4 p.m. and 6 p.m.

Rheumatic Diseases Conference

The 7th International Congress of Rheumatic Diseases will be held in New York City on May 30—June 3, 1949, under the auspices of the International League against Rheumatism. The invited guests will be the members of the International League, the European League, and the Pan-American League against Rheumatism with their constituent organizations, the Canadian Rheumatism Association, the British Empire Rheumatism Council, the Heberden Society of London, the Rheumatism Societies affiliated to the American Rheumatism Association, and certain individuals. Prospective participants who intend to present papers should submit titles of their papers to the Programme Committee before Jan. 1, 1949. Information may be obtained from Dr. Ralph Pemberton, 1901, Walnut Street, Philadelphia, Pennsylvania, U.S.A.

SOCIETIES AND LECTURES

Tuesday

EDINBURGH POSTGRADUATE BOARD FOR MEDICINE.—At Edinburgh Royal Infirmary (West Medical Lecture Theatre), Dec. 7, 5 p.m. *"A Scholar's View of Immortality,"* by Professor O. L. Richmond.

EUGENICS SOCIETY.—At Royal Society, Burlington House, Piccadilly, London, W., Dec. 7, 5.30 p.m. *"Maternity in Britain: The Results of a Social Survey,"* by Dr. J. W. B. Douglas. All interested are invited to attend.

GLASGOW UNIVERSITY.—Dec. 7, 4.30 p.m. *"Medicine in Literature,"* by Dr. Douglas Guthrie.

INSTITUTE OF DERMATOLOGY, 5, Lisle Street, Leicester Square, London, W.C.—Dec. 7, 5 p.m. *"Hysteria Cutis,"* by Dr. W. J. O'Donovan.

INSTITUTE OF UROLOGY.—At St. Paul's Hospital, Endell Street, London, W.C., Dec. 7, 11 a.m. *"Interpretation of Serological Reactions for Syphilis,"* by Dr. R. Thomson; at St. Peter's Hospital, Henrietta Street, London, W.C., Dec. 7, 5 p.m. *"Non-gonococcal Urethritis,"* by Dr. A. H. Harkness.

ROYAL COLLEGE OF PHYSICIANS OF LONDON, Pall Mall East, S.W.—Dec. 7, 5 p.m. *"The Pestilences of War—I, The Early Civilizations and Greece,"* FitzPatrick Lecture by Dr. W. H. Wynn.

ROYAL INSTITUTE OF PUBLIC HEALTH AND HYGIENE, 28, Portland Place, London, W.—Dec. 7, 3 p.m. *"The Physiology of Haemopoiesis,"* Harben Lecture by Sir Lionel Whitby.

Wednesday

BRITISH PSYCHOLOGICAL SOCIETY.—At Royal Society of Medicine, 1, Wimpole Street, London, W., Dec. 8, 8 p.m. Meeting of Medical Section. *"The Changing Functions of a Psychiatric Outpatient Department."* Symposium by Drs. Elizabeth Rosenberg, O. W. S. FitzGerald, and Sir Paul Mallinson.

INSTITUTE OF UROLOGY.—At St. Paul's Hospital, Endell Street, London, W.C., Dec. 8, 11 a.m. *"Biological False-positive Serum Reactions,"* by Dr. R. Thomson; 5 p.m., *"Fistula, Carcinoma, and Foreign Bodies of the Urethra, and Injuries of the Urethra,"* by Mr. C. H. Mills.

PHYSICAL SOCIETY: COLOUR GROUP.—At Large Physics Lecture Theatre, Imperial College, Imperial Institute Road, London, S.W., Dec. 8, 3 p.m. 43rd Science Meeting of Group. *"The Scotopic Visibility Function,"* by Dr. B. H. Crawford. *"Visual Purple and the Photopic Luminosity Curve,"* by Dr. H. J. A. Dartnall.

ROYAL FACULTY OF PHYSICIANS AND SURGEONS OF GLASGOW, 242, St. Vincent Street, Glasgow.—Dec. 8, 5 p.m. *"Poliomyelitis,"* by Professor J. C. Spence.

ROYAL INSTITUTE OF PUBLIC HEALTH AND HYGIENE, 28, Portland Place, London, W.—Dec. 8, 3 p.m. *"Dyshaemopoiesis from Nutritional and Specific Deficiencies,"* Harben Lecture by Sir Lionel Whitby.

ROYAL SANITARY INSTITUTE, 90, Buckingham Palace Road, London, S.W.—Dec. 8, 2.30 p.m. *"Food Hygiene: Ideas for the Future."* Discussion to be opened by Councillor William L. Roots and Mr. H. W. Walters.

UNIVERSITY COLLEGE, Gower Street, London, W.C.—At Physiology Theatre, Dec. 8, 5.15 p.m. *"Statistics from the Standpoint of a Pharmacologist,"* by Dr. J. W. Trevan, F.R.S.

Thursday

INSTITUTE OF DERMATOLOGY, 5, Lisle Street, Leicester Square, London, W.C.—Dec. 9, 5 p.m. *"Seborrhoeic Dermatoses,"* by Dr. J. L. Franklin.

INSTITUTE OF UROLOGY.—At St. Paul's Hospital, Endell Street, London, W.C., Dec. 9, 11 a.m. *"Treatment of Syphilis,"* by Dr. W. N. Mascall; 5 p.m., *"Injuries and Diseases of the Penis, other than Venereal,"* by Mr. A. R. C. Higham.

PLANNING FORUM.—At Planning Centre Hall, 28, King Street, Covent Garden, London, W.C., Dec. 9, 6.15 p.m. *"Manpower,"* discussion to be opened by Mr. A. E. U. Maud and Mr. G. D. N. Worswick.

ROYAL COLLEGE OF PHYSICIANS OF LONDON, Pall Mall East, S.W.—Dec. 9, 5 p.m. *"The Pestilences of War—II, The Roman Republic and Empire,"* FitzPatrick Lecture by Dr. W. H. Wynn.

ROYAL COLLEGE OF SURGEONS OF ENGLAND, Lincoln's Inn Fields, London, W.C.—Dec. 9, 5 p.m. *"The Reactions of Bone to Metal,"* Robert Jones Lecture by Sir Reginald Watson-Jones.

ROYAL INSTITUTE OF PUBLIC HEALTH AND HYGIENE, 28, Portland Place, London, W.—Dec. 9, 3 p.m. *"Dyshaemopoiesis from Noxious Agents,"* Harben Lecture by Sir Lionel Whitby.

ROYAL PHOTOGRAPHIC SOCIETY OF GREAT BRITAIN: SCIENTIFIC AND TECHNICAL GROUP, 16, Prince's Gate, London, S.W.—Dec. 9, 7 p.m. *"The Action of X Rays and Electrons on the Photographic Emulsion,"* by Dr. R. H. Herz.

ROYAL SOCIETY OF TROPICAL MEDICINE AND HYGIENE.—At 26, Portland Place, London, W., Dec. 9, 7.30 p.m. *"A Survey of Physiological Studies of Mental and Physical Work in Hot and Humid Environments,"* by Professor G. Crowden. A discussion will follow.

ST. GEORGE'S HOSPITAL MEDICAL SCHOOL, Hyde Park Corner, London, S.W.—Dec. 9, 4.30 p.m. *"Neurology and Psychiatry."* Lecture-demonstration by Dr. Desmond Curran.

SOCIETY OF CHEMICAL INDUSTRY: FINE CHEMICALS GROUP.—At London School of Hygiene and Tropical Medicine, Keppel Street, London, W.C., Dec. 9, 7 p.m. *"The Chemistry of Plant Growth Regulators,"* by Dr. W. A. Sexton.

Friday

LONDON CHEST HOSPITAL, Victoria Park, E.—Dec. 10, 5 p.m. *"Mediastinal Tumours,"* by Dr. J. Smart.

MAIDA VALE HOSPITAL MEDICAL SCHOOL, Maida Vale, London, W.—Dec. 10, 5 p.m. Case demonstration by Mr. Valentine Logue.

ROYAL MEDICAL SOCIETY, 7, Melbourne Place, Edinburgh.—Dec. 10, 8 p.m. Hat night.

WEST KENT MEDICO-CHIRURGICAL SOCIETY.—At Miller Hospital, Greenwich High Road, London, S.E., Dec. 10, 8.30 p.m. *"New Chemotherapeutic Substances of Microbial Origin,"* Purvis Oration by Sir Howard Florey, F.R.S.

No. 46

INFECTIOUS DISEASES AND VITAL STATISTICS

We print below a summary of Infectious Diseases and Vital Statistics in the British Isles during the week ended Nov. 13.

Figures of Principal Notifiable Diseases for the week and those for the corresponding week last year, for: (a) England and Wales (London included). (b) London (administrative county). (c) Scotland. (d) Eire. (e) Northern Ireland.

Figures of Births and Deaths, and of Deaths recorded under each infectious disease, are for: (a) The 126 great towns in England and Wales (including London). (b) London (administrative county). (c) The 16 principal towns in Scotland. (d) The 13 principal towns in Eire. (e) The 10 principal towns in Northern Ireland.

A dash — denotes no cases; a blank space denotes disease not notifiable or no return available.

Disease	1948 (a)	(b)	(c)	(d)	(e)	1947 (Corresponding Week) (a)	(b)	(c)	(d)	(e)
Cerebrospinal fever	33	7	12	3	—	40	2	26	1	—
Deaths		2	2				—			
Diphtheria	159	18	57	6	5	195	27	60	25	5
Deaths	2	—	—	—	—	2	1	2	—	1
Dysentery	133	19	53	—	—	95	9	17	—	—
Deaths				—	—				—	—
Encephalitis lethargica, acute	2	—	1	—	—	2	—	—	—	—
Deaths		—					—			
Erysipelas			37	13	1			48	16	4
Deaths		—					—			
Infective enteritis or diarrhoea under 2 years				54					53	
Deaths	32	2	7	2	3	61	4	13	2	6
Measles*	6,910	108	119	50	46	2,355	116	224	227	1
Deaths†			—	—	—	2	—	—	—	1
Ophthalmia neonatorum	45	7	6	—	—	58	5	12	—	—
Deaths										
Paratyphoid fever	8	2	17 (B)	—	—	8	—	—	—	—
Deaths	—	—	—	—	—	—	—	—	—	—
Pneumonia, influenzal	579	39	13	3	2	582	41	8	1	13
Deaths (from influenza)‡	14	2	2	2	—	13	2	—	—	—
Pneumonia, primary			200	24				321	21	
Deaths	202	42		6	7		49		9	7
Polio-encephalitis, acute	3	—				15	2			
Deaths							1			
Poliomyelitis, acute	60	3	3	3	—	186	12	18	7	—
Deaths§	4	1					—			
Puerperal fever		1	5		1		—	13		—
Deaths										
Puerperal pyrexia‖	92	7	5	—	—	117	4	7	—	4
Deaths		1					—			
Relapsing fever	—	—			—	—	—			—
Deaths										
Scarlet fever	1,450	97	302	207	38	1,947	124	353	54	42
Deaths†			—	—	—	3	1	—	—	—
Smallpox	—	—	—	—	—	—	—	—	—	—
Deaths				—	—				—	—
Typhoid fever	5	—	1	—	—	11	3	—	2	3
Deaths	1	—	—	—	—	—	—	—	—	—
Typhus fever	—	—	—	—	—	—	—	—	—	—
Deaths				—	—				—	—
Whooping-cough*	2,481	152	101	56	10	1,217	70	43	36	7
Deaths	6	—	—	—	—	3	—	—	—	1
Deaths (0–1 year)	279	35	42	22	12	378	47	51	32	20
Infant mortality rate (per 1,000 live births)										
Deaths (excluding stillbirths)	4,876	801	658	171	118	4,582	742	593	179	118
Annual death rate (per 1,000 persons living)			13·3	10·7				12·3	11·3	
Live births	7,065	1124	851	417	180	7,781	1260	907	312	224
Annual rate per 1,000 persons living			17·2	26·1				18·3	19·7	
Stillbirths	179	20	30			196	28	29		
Rate per 1,000 total births (including stillborn)			34					31		

* Measles and whooping-cough are not notifiable in Scotland, and the returns are therefore an approximation only.

† Deaths from measles and scarlet fever for England and Wales, London (administrative county), will no longer be published.

‡ Includes primary form for England and Wales, London (administrative county), and Northern Ireland.

§ The number of deaths from poliomyelitis and polio-encephalitis for England and Wales, London (administrative county), are combined.

‖ Includes puerperal fever for England and Wales and Eire.

EPIDEMIOLOGICAL NOTES

Discussion of Table

In *England and Wales* an increased incidence was recorded for measles 733, whooping-cough 241, acute pneumonia 83, and dysentery 78.

The increase in the notifications of measles was due to a few counties; in the remainder of the country only slight variations occurred. The largest increases were Lancashire 390, Yorkshire East Riding 107, Derbyshire 93, and Suffolk 54.

The largest rises in the incidence of whooping-cough were 80 in Lancashire and 57 in Wales. The rise in Wales was due mainly to an outbreak affecting 37 persons in the rural districts of Twrcelyn and Valley in Anglesey.

The chief feature of the returns of diphtheria was an outbreak of 12 cases in Yorkshire West Riding, Hoyland Nether U.D. No large fluctuations in the notifications of scarlet fever were recorded. The largest rise in the incidence of acute pneumonia was 32 in Yorkshire West Riding.

No further case of typhoid was notified in Shropshire, Oswestry R.D., where the outbreak which had persisted for the preceding nine weeks had resulted in 105 notifications.

Fresh outbreaks of dysentery during the week were notified from Essex, Hornchurch U.D. 60, and Cornwall, Truro R.D. 7. An increase of 12 in the cases of dysentery was reported from London, where the notifications were distributed over nine boroughs. The only other large return of dysentery was Lancashire 19.

Acute poliomyelitis declined from 73 to 60 notifications; the largest returns were Durham 7, Lancashire 4, and Middlesex 4.

In *Scotland* rises occurred in the notifications of acute primary pneumonia 13, whooping-cough 12, and paratyphoid fever 11, while decreases were recorded for measles 47 and scarlet fever 12. All the 17 cases of paratyphoid fever were notified in the city of Aberdeen. The chief centres of dysentery were the cities of Glasgow 31 and Edinburgh 14.

In *Eire* decreases in the notifications of measles 74 and whooping-cough 16 were the chief features of the returns. A fall of 11 was recorded in the notifications of diarrhoea and enteritis in Dublin C.B.

In *Northern Ireland* the notifications of measles in Belfast C.B. were 29 fewer than in the preceding week.

Week Ending November 20

The notifications of infectious diseases in England and Wales during the week included: scarlet fever 1,835, whooping-cough 2,680, diphtheria 154, measles 6,923, acute pneumonia 732, cerebrospinal fever 32, acute poliomyelitis 46, dysentery 49, paratyphoid 4, and typhoid 9.

BIRTHS, MARRIAGES, AND DEATHS

BIRTHS

Calvert-Smith.—On Sept. 18, 1948, to Daphne (née Bousfield), M.B., B.S., wife of John E. Calvert-Smith, M.A., of 60, Hall Farm Drive, Whitton, Middlesex, a son—Colin Paul.

Cane.—On Nov. 17, 1948, at Tabora, Tanganyika, to Margaret, wife of Dr. Hugh Cane, a son.

DEATHS

Bonavia.—On Nov. 20, 1948, at No. 94, British Military Hospital, Hamburg, Victor Joseph Bonavia, M.D., M.R.C.P., Colonel, late R.A.M.C., aged 54.

Deuchars.—On Nov. 20, 1948, at 38, Hope Terrace, Edinburgh, James McGavin Deuchars, M.B., Ch.B.Ed.

Hamilton.—On Nov. 12, 1948, at 205, East 61st Street, New York, George Johnson Hamilton, M.D.

Heath.—On Nov. 16, 1948, at 9, Highdown Avenue, Worthing, Wroughton Gerald Heath, M.B., Ch.B.Ed., aged 75.

Kitchin.—On Nov. 22, 1948, Percy Kitchin, M.R.C.S., L.R.C.P., of 86, Sea Lane, Goring-by-Sea, aged 78.

Maples.—On Nov. 16, 1948, at 7, Windsor Crescent, Jersey, Ernest Edgar Maples, M.D., F.R.C.S.

Moseley.—On Nov. 21, 1948, at Ipswich, Charles Kingdon Moseley, M.R.C.S., L.R.C.P., aged 84.

Oakman.—On Nov. 22, 1948, at a nursing-home in Hove, Joseph John Oakman, M.R.C.S., of Rutlands, Upper Shoreham Road, Shoreham-by-Sea, Sussex, aged 84.

Palmer.—On Nov. 22, 1948, at Morningside, Prestbury, near Cheltenham, Harold Thornbury Palmer, M.R.C.S., L.R.C.P., D.D.M.S., Nigeria, retired.

Picton.—On Nov. 19, 1948, at Holmes Chapel, Cheshire, Lionel James Picton, O.B.E., B.M., B.Ch., aged 74.

Roberts.—On Nov. 20, 1948, at Northbrook, Pangbourne, Berks, Harry Trist Roberts, L.M.S.S.A., L.D.S., R.C.S., late of 31, Harley Street, London, W.

Robertshaw.—On Nov. 21, 1948, Walter Midgley Robertshaw, M.B., C.M.Ed., of Knoll Top, Stocksbridge, Sheffield, aged 78.

Rozelaar.—On Nov. 21, 1948, at 3, Gillingham Road, London, N.W., Abraham Levie Rozelaar, M.R.C.S., L.R.C.P.

Waddy.—Recently, Frederick Henry Waddy, M.D.Glas., formerly of Sheffield, aged 79.

Wallington.—On Nov. 17, 1948, at sea, Kenneth Tratman King Wallington, M.R.C.S., L.R.C.P.

Any Questions?

Correspondents should give their names and addresses (not for publication) and include all relevant details in their questions, which should be typed. We publish here a selection of those questions and answers which seem to be of general interest.

Classification of Pulmonary Tuberculosis

Q.—*What is the classification of pulmonary tuberculosis of the National Tuberculosis Association of America, and how does this compare with the latest Ministry of Health classification in this country?*

A.—The classification of pulmonary tuberculosis of the National Tuberculosis Association of America was revised in 1940, when the terms "primary phase" and "reinfection phase" replaced "childhood type" and "adult type." The features of previous classification relating to extent of lesions and symptomatology were preserved, but definitions were made more comprehensive and those relating to results of treatment were more rigidly specified. The definitions and classifications may be briefly summarized as follows.

Primary Phase.—That phase of the morbid process which follows directly and uninterruptedly the first implantation of tubercle bacilli. It may occur at any age and may be symptomless. There may be no clinical or radiological signs, but, on the other hand, the infection may be grave.

Reinfection Phase.—This follows the primary phase usually after a latent period, and comprises the lesions which develop after reinfection with tubercle bacilli, which are derived from five potential sources: (*a*) outside the body; (*b*) from a breakdown of the primary focus; (*c*) by rupture of a caseous lymph node by way of the lymphatics and the blood stream; (*e*) from the small apical foci resulting from early haematogenous dissemination becoming progressive after having been stationary or undetected for a long period of time.

Pulmonary Lesions.—These are classified as: (1) *Minimal.*—Slight lesions without demonstrable excavation confined to a small part of one or both lungs; the total extent of the lesions, regardless of distribution, shall not exceed the equivalent of the volume of lung tissue which lies above the second chondro-sternal junction and the spine of the fourth or body of the fifth thoracic vertebra on one side. (2) *Moderately advanced.*—One or both lungs may be involved, but the total extent of the lesions shall not exceed the following limits: (*a*) slight disseminated lesions which may extend through not more than the volume of one lung; (*b*) dense and confluent lesions which may extend through not more than the equivalent of one-third the volume of one lung; (*c*) any gradation within the limits of *a* and *b*. Total cavities, if present, should not exceed 4 cm. (3) *Far advanced.*—Lesions more extensive than moderately advanced.

Clinical Status.—Under this heading five definitions are stated:

(1) *Apparently Cured.*—In which constitutional symptoms are absent and sputum, if any, must be negative for tubercle bacilli, not only by concentration and microscopical examination, but also by culture and animal inoculation. In case there is no sputum, the fasting gastric contents should be obtained and similarly examined. Lesions must be stationary and apparently healed according to *x*-ray examination. These conditions shall have existed for a period of two years under ordinary conditions of life. (2) *Arrested.*—Constitutional symptoms absent; sputum, if any, must when concentrated be microscopically negative for tubercle bacilli; lesions stationary and apparently healed according to *x*-ray examination; no evidence of pulmonary cavitation. These conditions shall have existed for a period of six months, during the last two of which the patient has been taking one hour's walking exercise twice daily or its equivalent. (3) *Apparently Arrested.*—Clinical conditions demanded for "arrested" shall have existed for a period of three months, during the last two of which the patient has been taking one hour's walking exercise daily or its equivalent. (4) *Quiescent.*—No constitutional symptoms; sputum, if any, may contain tubercle bacilli; lesions stationary or retrogressive according to *x*-ray examination; cavity may be present. These conditions to have existed for at least two months, during which time the patient has been ambulant. (5) *Frankly Active.*—Improved, unimproved. Symptoms unchanged, worse, or less severe, but not completely abated. Lesions not completely healed or progressive according to *x*-ray examination. Sputum almost always contains tubercle bacilli. (6) *Dead.*

The U.S. classification has had to be given in considerable detail, otherwise it would be difficult to point out how that of the Ministry of Health differs from it. The fundamental difference between the Ministry's classification and the American one is that the former is based on the finding, or failure to find, the tubercle bacillus in the lesion, thereby dividing tuberculous persons into two classes: (*a*) the non-infectious, in whom the tubercle bacillus has never been found; and (*b*) the infectious class, in whom the bacillus has been found. Both classes are subdivided into three groups (1, 2, and 3) according to the radiological extent of the lesion and the severity of the symptoms. Cases of pleural effusion in which there is an absence of severe constitutional disturbances, failure to find tubercle bacilli in the pleural fluid, and no parenchymatous lesion are placed in Group 1 of Class A. Where a single positive bacteriological report is not confirmed by further bacteriological search and is unsupported by clinical or radiological evidence of tuberculosis it may be ignored. Considerable difference exists in the definitions of "quiescence." The one adopted by the Ministry of Health reads:

"Cases in which the general condition and exercise tolerance are good, having regard to the extent of the lesion; which show no evidence of toxaemia; in which no tubercle bacilli have been found on three consecutive monthly examinations by stained film; and in which changes revealed by other clinical investigations and by serial skiagrams point to retrogression of the tuberculous lesion."

A case is not accepted as "recovered" until a state of quiescence has been maintained without interruption for five years. The term "active" is described by a negative definition as "the state that is not quiescent." All cases discharging tubercle bacilli within the preceding three months should be considered as "active." A stationary case is one in which the signs, symptoms, clinical tests, and radiological appearance of the lesion have presented no material new feature during the period under review.

To draw up a classification which will satisfactorily include all cases of tuberculosis is a difficult task, and many attempts have been made to produce a comprehensive scheme, but none up to the present meets all requirements. Both the one of the National Tuberculosis Association and that of the Ministry of Health are practical classifications with limited application, but are useful and satisfactory for statistical work on an extensive scale.

Leukoplakia and Kraurosis Vulvae

Q.—*What are the present definitions of leukoplakia and kraurosis vulvae? Which is the commoner? What are the modern figures for the relationship between each of these two conditions and malignancy?*

A.—Although the general opinion on these two conditions is still based on the views put forward by Berkeley and Bonney in 1909 (*British Medical Journal*, 1909, **2**, 1739; *Proc. R. Soc. Med.*, 1909, **3**, 29) there remains a good deal of doubt and confusion. Indeed, there are signs of a reorientation of ideas during the last few years, and this tends to increase the uncertainty. Kraurosis vulvae is to be regarded as a condition of extreme atrophy and contracture of the introitus, sometimes complicated by secondary infection. It might be considered as an exaggeration of the normal senile atrophy, and for all practical purposes it occurs only after suppression of ovarian activity. It is conceivable, however, that it might arise before the menopause if for some reason the vulva loses its ability to respond to oestrogens. In the typical menopausal case the lesion responds to oestrogens but recurs when they are withheld. It does not predispose to cancer. Its occurrence is rare as compared with that of leukoplakia.

Leukoplakia vulvae is characterized by excessive keratinization of the skin, which becomes thickened, white, and hard and tends to crack, and it is not strictly confined to the introitus. In the deeper layers of the dermis, in the later stages of the disease at any rate, there are degenerative changes, and the appearances suggest that the nutrition or blood supply of the epidermis is impaired. It is doubtful whether the four separate pathological stages of the lesion as described by Berkeley and Bonney really exist. It was formerly thought to be a definite clinical entity of unknown cause, possibly a manifestation of chronic infection, but not syphilitic. There is now a good deal of

evidence to suggest that the skin changes represent a non-specific reaction to a large number of possible factors. Among these factors deficiency states may be important, so "leukoplakia" is sometimes seen as a manifestation of riboflavin deficiency, iron-deficiency anaemia, achlorhydria, or macrocytic anaemia, and clears up when the missing factor is replaced. Sometimes, perhaps, the deficiency is a local rather than a general one, and results from an interference in blood supply to the vulval skin or from a failure of the tissues to utilize the factor concerned. Scratching by the patient in response to pruritus vulvae of any origin may also account for some of the skin changes. Leukoplakia can occur at any age and is probably not related to failure of ovarian function; it is not improved by oestrogen therapy. These are some of the lines of modern thought; the ideas are attractive, but many await proof.

Unlike kraurosis, leukoplakia is a precancerous condition, and that at any age. It is impossible to give reliable statistics about how often cancer is superimposed, but it is frequent enough to justify excising areas of leukoplakia which do not respond readily to medical treatment. There are figures showing that established carcinoma of the vulva is very frequently associated with leukoplakia in adjacent areas of skin, suggesting that leukoplakia preceded the growth. Berkeley and Bonney (*loc. cit.*) said they had never seen a case of carcinoma without there being evidence of leukoplakia as well. F. J. Taussig, the American authority on this group of diseases, found leukoplakia to be present in 50% of cases, while S. Way (*Ann. roy. Coll. Surg. Eng.*, 1948, **3**, 187), in his recent report on a comparatively large series of cases of cancer of the vulva, puts the figure at 78%.

Fattening the Lean

Q.—*Is there any treatment which will increase the weight of very thin people who appear to have no other abnormalities? A man of 30 weighs 9 st. 7 lb. (60.3 kg.) and is 6 feet (1.8 m.) tall; he has been this weight for twelve years. An office worker, he is very fit but tires rather easily. He has no relevant previous history and his diet seems to be adequate. He asks if insulin injections would be helpful.*

A.—The problem of increasing the weight of the abnormally thin is complementary to that of reducing the weight of the abnormally fat. Assuming the absence of morbid causes, in the latter the intake of calories exceeds the output; in the former, the reverse. Many believe that constitutional factors are important: that in some, absorption is unduly efficient or utilization greater. No evidence exists to suggest that these views are correct. In the absence of disease the tendency to gain or lose weight appears to be a simple function of the quantity of food eaten and the amount of energy expended in work. It depends, that is, on appetite, and appetite is a habit without close correlation to the energy output of the body. While those who gain weight always eat more than they need, it is proverbial that they invariably claim to eat "hardly a thing." Similarly, careful analysis of the exact quantities eaten by those who are unduly thin will show that the calorie intake is low. They will seldom agree that they eat insufficient, and they do, indeed, take enough to assuage their small appetites. It will be found that if a diet of sufficient calorie value is taken the thin will certainly gain weight.

In the present instance the mean normal weight for a man of 30 years, 6 feet tall, is 12 st. 4 lb. (78 kg.). He is thus 39 lb. (17.6 kg.) under weight. The estimated basal requirements of a normal man of this age and height would be 1,920 calories; average requirements for a moderately active life are a little above twice the basal values. If he took a diet of 3,800 calories there is little doubt that his weight would rise to normal levels. Insulin has the effect of increasing appetite, and might be of value in this respect.

Phosphaturia

Q.—*What is the best treatment for phosphaturia? What measures should be taken to counteract it?*

A.—Phosphates are, of course, present in normal urine, and it is quite usual for phosphatic salts to be deposited when the urine cools. Sometimes phosphates come out of solution before the urine leaves the bladder, and the urine is then cloudy when passed. This is not indicative of any state of ill-health and has not been shown to predispose towards the formation of calculi. It therefore requires no treatment, but if it worries the patient it can be prevented by acidifying or diluting the urine. This could be done by taking more fluid and a diet containing more protein and less fruit and vegetables. Acidifying salts such as ammonium chloride would have the same effect, but there is no reason to give these unpleasant remedies for this harmless symptom.

Quinidine, Dicoumarol, and Auricular Fibrillation

Q.—*In view of the serious consequences which may follow embolism in cases of auricular fibrillation treated with one of the digitalis compounds, is it justifiable to give a course of dicoumarol at the onset of the digitalis therapy? If so, for how long and in what dosage? Does digitalis act on the blood by increasing the clotting power, or is it simply that thrombi are more likely to be discharged from the auricles as a result of the altered action of the heart under the influence of digitalis?*

A.—Digitalis compounds do not in themselves increase the likelihood of embolic phenomena when given in cases of auricular fibrillation. Digitalis increases coagulability of the blood to such a slight degree that it has no clinical significance. In the case of quinidine, which apparently is the drug in mind, embolic phenomena may follow its use. This danger depends on the presence of pre-formed thrombi in the auricular appendages, which are expelled when the auricles revert to sinus rhythm. Its danger is not removed by a course of dicoumarol, but may be avoided in part by the careful selection of cases for quinidine therapy. This drug should not be given to a patient known to be fibrillating for eighteen months or longer, who has been in congestive failure, who has already suffered embolic accidents, or who has severe cardiac damage.

NOTES AND COMMENTS

Herpes after Coryza.—Dr. M. A. DOBBIN CRAWFORD (London, W.C.) writes: I was interested in the question and answer under this heading ("Any Questions?" Oct. 16, p. 730). I am convinced that in the majority of cases this condition is due to superficial irritation of the skin about the nose and lips by the catarrhal nasal discharge—very probably infective. The typical blister reaction can be completely prevented by the use of a suitable barrier cream from the start of the cold. The best I have found is a sulphacetamide ointment. If applied freely at night and when possible during the day there will be no herpes. In cases where the skin is already blistered it should be dried and cleaned by frequent application of spirits of camphor, eau de cologne, or methylated spirits, followed by the use of the sulphacetamide ointment.

Physiology of Hearing.—Dr. R. S. CREED (Oxford) writes: The answer (Nov. 6, p. 846) to the request for information about the specific gravity and viscosity of the cochlear fluids may be supplemented as follows. In both shark (Kaieda, J., *Hoppe-Seyl. Z.*, 1930, **188**, 193) and pigeon (Rossi, G., *Arch. Fisiol.*, 1914, **12**, 415) endolymph has been found to be much more viscous than perilymph. In the shark they have the same specific gravity (1020), slightly lower than that of cerebrospinal fluid (1023). Other details of chemical and physical properties are given by Kaieda, and by Gerhartz in Oppenheimer's *Handbuch der Biochemie* (1925, **4**, 182. Jena).

Christmas at Barnardo's.—In Dr. Barnardo's Homes 7,000 children are making plans for Christmas. The Homes would gratefully receive gifts of toys or money, which should be sent to Barnardo House, Stepney Causeway, London, E.1.

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